STUDY OF SEROPREVALENCE OF HEPATITIS B VIRUS IN MEDICO LEGAL AUTOPSIES

Dissertation submitted in partial
fulfillment of the requirements for the
degree

M.D. (Forensic Medicine)

BRANCH - XIV

INSTITUTE OF FORENSIC MEDICINE

MADRAS MEDICAL COLLEGE

CHENNAI – 600 003

THE TAMIL NADU

Dr. M.G.R. MEDICAL UNIVERSITY

CHENNAI

APRIL 2015

BONAFIDE CERTIFICATE

This is to certify that the work embodied in this dissertation entitled **"STUDY OF SEROPREVALENCE OF HEPATITIS B VIRUS IN MEDICO LEGAL AUTOPSIES"** has been carried out by **Dr. S.Ramalingam** a Post Graduate student under my supervision and guidance for his study leading to Branch XIV M.D. Degree in Forensic Medicine during the period of April 2014 to September 2014

DEAN
Madras Medical College & Medicine
Rajiv Gandhi Govt. General Hospital
Chennai-3

Date:

Place:

Director and Professor
Institute of Forensic Medicine
Madras Medical College
Chennai-3

Date:

Place:

DECLARATION

I, **Dr. S. Ramalingam**., solemnly declare that this dissertation titled **"STUDY OF SEROPREVALENCE OF HEPATITIS B VIRUS IN MEDICO LEGAL AUTOPSIES"** is the bonafide work done by me under the expert guidance and supervision of **Prof.Dr.R.Vallinayagam**, Director and Professor, Institute of Forensic Medicine, Madras Medical College, Chennai – 3. This dissertation is submitted to The Tamil Nadu Dr. M.G.R Medical University towards partial fulfillment of requirement for the award of M.D., Degree (Branch XIV) in Forensic Medicine.

Place: **Dr. S. Ramalingam**

Date:

ACKNOWLEDGEMENT

I am greatly obliged to the Dean, **Dr.Vimala** MD, Madras Medical College and Rajiv Gandhi Govt. General hospital, Chennai -3 for allowing me to conduct this study.

I am especially thankful to my Director and Professor **Dr.R.Vallinayagam MD**, Associate Professor **Dr. M.N. Rajamani Bheem Rao MD** and Assistant Professor **Dr. T. Vedanayagam MD**, Institute of Forensic Medicine, Madras Medical College, Chennai -3 for their interest and encouragement, in bringing out this dissertation for my MD exam.

I would like to express my heartfelt gratitude to my esteemed Professor & Former Director, **Capt. Dr B. Santhakumar MD**, who has been a constant source of inspiration and encouragement.

I am thankful to **Dr. Vindhu**, Director Winpath Labs, Mylapore, who guided me in every possible way in doing my thesis.

I thank all my Colleagues for helping, in collecting material for my study.

INSTITUTIONAL ETHICAL COMMITTEE

APPROVAL LETTER

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CERTIFICATE OF APPROVAL

To

Dr.S.Ramalingam,
Postgraduate
Institute of Forensic Medicine,
Madras Medical College, Chennai-3.

Dear **Dr.S.Ramalingam,**

The Institutional Ethics Committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled **"Study of Seroprevelance of Hepatitis B Virus in Medicolegal Autopsies"** No.12042014.

The following members of Ethics Committee were present in the meeting held on 08.04.2014 conducted at Madras Medical College, Chennai-3.

1. Dr. C.Rajendran, M.D, -- Chairperson
2. Prof. Kalaiselvi, M.D, -- Member Secretary
   Vice Principal, MMC, Ch-3
3. Prof. Nandhini, M.D, -- Member
   Inst. of Pharmacology, MMC, Ch-3
4. Prof.Bhavani Sankar, M.S, -- Member
   Prof & HOD General Surgery, MMC, Ch-3
5. Prof.V.Padmavathi, M.D, -- Member
   I/c. Director of Pathology, MMC, Ch-3
6. Thiru. S. Govindasamy, BA., BL -- Lawyer
7. Tmt.Arnold Saulina, MA MSW -- Social Scientist
8. Thiru.S.Ramesh Kumar, -- Lay Person
   Administrative Officer, MMC, Ch-3.

We approve the proposal to be conducted in its presented form.

Sd/Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

Member Secretary, Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
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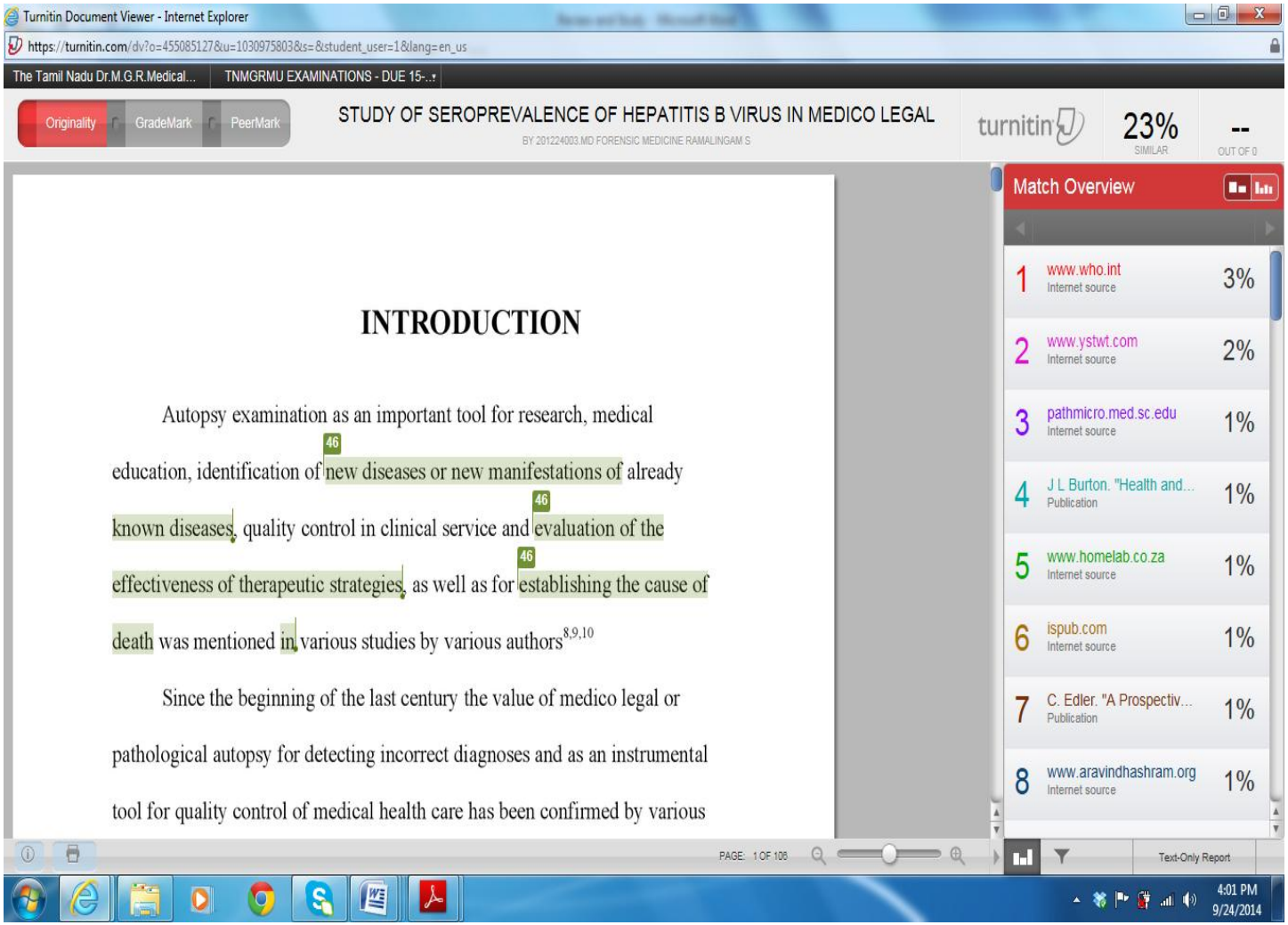
PLAGIARISM PERCENTAGE



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LIST OF ABBREVIATIONS

AB = Antibody

AFP = Alpha foeto protein

Ag = Antigen

AIDS = Acquired Immunodeficiency syndrome

ALAT = Alanine aminotranferase

CDC = Centers for Disease Control

ELISA = Enzyme linked Immunosorbent assay

HAV = Hepatitis A Virus

HBV = Hepatitis B Virus

HBcAg= Hepatitis B core antigen

HBcAb = Hepatitis B core Antibody

HbeAg= Hepatitis B envelope antigen

HbeAb=Hepatitis B envelope antibodies

HBsAg = Hepatitis B Surface Antigen

HCC = Hepato cellular carcinoma

HCV = Hepatitis C Virus

HDV = Hepatitis D Virus

HIV = Human Immunodeficiency Virus

HPE = HistoPatholical Examination

IgG = Immunoglobulin G

IgM = Immunoglobulin M

IHC = Immuno histo chemistry

NACP= National AIDS Control Program

PCR= polymerase chain reaction

US = United States

WHO= World Health Organization

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ABSTRACT FOR THE DISSERTATION TO BE SUBMITTED TO

THE TAMILNADU Dr M.G.R MEDICAL UNIVERSITY, CHENNAI

FOR APRIL 2015 M.D.FORENSIC MEDICINE EXAMINATIONS

TITLE: STUDY OF THE SERO - PREVALENCE OF HEPATITIS B VIRUS IN MEDICO LEGAL AUTOPSIES

Abstract:

Medico - legal practice is associated with a significantly increased risk when compared to other medical specialties, both in terms of airborne and blood borne diseases. However, the availability of data regarding these potential occupational risks is limited. Safety becomes an issue, both in medical and ecological aspects regarding the protection of environment with the high seroprevalence of HIV and hepatitis viruses. Hepatitis B virus has the highest transmissibility rate with a rate of about 100 times greater than HIV. Hepatitis B virus is associated with an increased morbidity and mortality among the mortuary workers due to its high frequency among the deceased and longevity of the virus. Till now only a few retrospective data about the post- mortem behavior of serological parameters of HBV, HCV and HIV parameters over time exist. The testing of postmortem sera for HbsAg will be a most reliable measure of antemortem HBV infection. HbsAg and anti -HbsAg have been shown to remain detectable in postmortem serum stored for relatively long periods of time. We conducted the Study to find the seroprevalence of HBV in routine autopsy cases.

Materials and methods: The study sample consists of 515 routine autopsy cases at Institute of Forensic Medicine, Madras Medical College. The samples were tested using a Human enzyme linked immunoassay kit for the presence of HBsAg.

Conclusion: Out of 515 cases, there are eighteen positive cases and thirteen positive cases were male, four positive cases were female and one case was transgender. The presence of 18 positive cases among 515 cases, though statistically insignificant is higher than many other voluntary screening programs. This shows that HBV screening is of great importance among the community. The present study concludes that testing of HBV in medico legal autopsies is a convenient and effective method in monitoring the surveillance of HBV-infection in the general population and it can be used for epidemiological studies. This screening test for the HBsAg may not be sensitive if the person was in the window period. It could be used along with unlinked anonymous tests from hospital and other similar patient materials.

Keywords: Hepatitis B, Viral hepatitis, Autopsy, HBsAg, Screening test

INTRODUCTION

Autopsy examination as an important tool for research, medical education, identification of new diseases or new manifestations of already known diseases, quality control in clinical service and evaluation of the effectiveness of therapeutic strategies, as well as for establishing the cause of death was mentioned in various studies by various authors[1,2,3]

Since the beginning of the last century the value of medico legal or pathological autopsy for detecting incorrect diagnoses and as an instrumental tool for quality control of medical health care has been confirmed by various studies[4,5,6].

Autopsies were conducted on an average of 50% of deaths in 1940s, but less than 10% in the 1990s, and these statistics were applied both at the level of universities and community hospitals, and in both the developed and most developing countries[7,8,9].

The decreasing numbers of medico legal autopsies have had many repercussions on the systematic errors and bias in the research data. As the data provided in the death certificates are the basis of epidemiology and health statistics, they need to be more accurate. Many decisions regarding public health are made based on incorrect or incomplete information they obtained from the hospital records about the main

diagnoses and causes of death, as in most of the cases it was established without any autopsy records[10].

Autopsy rates have fallen over the last few decades in spite of the overwhelming scientific evidence of the merits of the postmortem medical examination in modern medical practice[11, 12, 13]

Furthermore, it is the role of the autopsy surgeon to identify the dead body subjected for post mortem work and he has to arrive at the cause for the death by doing a detailed dissection of the tissues which are often in various stages of decomposition[14].

Despite the precautions taken to control the infection and availability of various vaccines, the health care professionals who had engaged themselves in the medical and medico legal practice are at risk of acquiring blood-borne viral infections by exposing them to body tissues or body fluids which are often loaded with infectious pathogens, irrespective of the stage of human remains.

AIMS AND OBJECTIVES

AIMS AND OBJECTIVES

Primary Objective: To study the Sero - Prevalence of Hepatitis B virus in Medico Legal Autopsies.

Secondary Objectives:

- To analyze the risk ratio and create awareness among the heath care personnel who handles dead bodies
- To diagnose the clinically undetected HBV cases.
- To determine whether postmortem of dead bodies which are thought to be at low risk groups, are safe or not.
- To formulate guidelines for a policy concerning HBV screening in forensic autopsy cases
- To identify the Sero prevalence of HBV in autopsy room personnel

REVIEW OF LITERATURE

HIGH-RISK AUTOPSY

In general, all medico legal or pathological autopsies are of high risk nature only. The infectivity status of the deceased person was not known in majority of the cases subjected for autopsy, as said before. It is well known that dissecting rooms and forensic laboratories are high-risk areas for acquiring infection, and forensic staffs have the risk of occupational exposure to these infectious agents, especially during post-mortem investigations.

Definition: The Postmortem examination of a deceased person who has had, or is likely to have had, a serious infectious disease that can be transmitted to those who present at the autopsy, thereby causing them serious illness and/or premature death[15].

The individuals who ignores, or ignorant of, the potential hazards at necropsy are liable not only to themselves but also to the colleagues working in or visiting to the mortuary or handling the dead bodies or biological material derived from it after autopsy.

An autopsy may subject the prosecutors and others to a wide variety of infectious agents, including blood borne and aerosolized pathogens such as human immunodeficiency virus, hepatitis B and C viruses, and Mycobacterium tuberculosis. Other hazards which are included in this are toxic chemicals (e.g., formalin, cyanide, and

organophosphates) and radiation from radio nuclides which are used for diagnosis and therapy [16].

Medico -legal practice is associated with a significantly increased risk when compared to other medical specialties, both in terms of airborne and blood borne diseases; several studies revealed the increased prevalence of hepatitis B, C, D, G, tuberculosis, HIV, prion diseases, hantavirus, measles, HTLV-1 or bacterial infections in mortuary workers[17.]

As infections caused by pathogens such as Mycobacterium tuberculosis, HBV, HIV etc are frequently asymptomatic when the deceased was alive and without morphological evidence but with retained infectivity when brought for necropsy[18].

However, the availability of data regarding these potential occupational risks is limited. Safety becomes an issue, both in medical and ecological aspects regarding the protection of environment with the high seroprevalence of HIV and hepatitis viruses[19].

The highest rate of laboratory- acquired air borne and blood borne infections from the dead bodies was seen in autopsy workers which was also established by the studies conducted between 1970 and 1989 in British clinical laboratories[20].

Airborne, contact routes and sharp instrument or needle injuries spread these infections from the corpses to workers [15,21,22,23] . However, in

many forensic situations the statistical risk of hepatitis and HIV infection are markedly greater than in the general autopsy population when they involve homosexuals and drug abusers[23].

HEPATITIS B VIRUS

Hepatitis B virus has a global distribution[24,25] .More than 240 million people have chronic (long-term) liver infections. About 600 000 people die every year due to the acute or chronic consequences of hepatitis B. The World Health Organization (WHO) considers hepatitis B virus (HBV) to be second to tobacco among the carcinogens[26]

Hepatitis B Virus, Hepatitis C Virus, as well as HIV, are the major sources of worldwide public health concern[15,21]. Amongst all the parenteral viruses, Hepatitis B virus has the highest transmissibility rate with a rate of about 100 times greater than HIV. It can lead to an acute infection, with a high chance of complete recovery or a latent infection with an increased risk of chronicity and hepatocellular carcinoma.

Hepatitis B virus is associated with an increased morbidity and mortality among the mortuary workers due to its high frequency among the deceased and longevity of the virus[14].HBV, HCV and HIV share similar modes of transmission and are relatively frequent among certain high- risk groups.

The increased duration in the profession increases the prevalence rate among the health care workers with 30% in those with 20 or more

years of duration in their profession and 5%in the general population in persons of comparable age[27].

When compared to physicians, the pathologists are considered as a high-risk group for occupationally acquired HBV infection due to their exposure to blood and body fluids[28].

Studies conducted in US found the highest risk for HBV infection was amongst surgeons and pathologists (about 6%)[28].

Study showed that the technicians having 8.8% of hepatitis B positivity due to his direct contact with blood while doing his professional work. Surveillance of health care workers or forensic medicine personnel suffering sharp injuries suggests that the presence of 'e' antigen (HBeAg) in the contaminating blood increases the overall risk of acquiring infection from 5% to 30%[29].

CDC also noticed the similar increased risk of acquiring infection from 5% to 30% if the blood is contaminated with HBe antigen[30]

The highest risk is noted among the nurses/ auxiliary personnel[31]; for example in Austr [p099ia there were identified the following relative risks: nurses – 30.6%, auxiliary personnel - 30.4%, physicians – 13.9%, laboratory personnel 2.9% and other 22.3%[32].

PREVIOUS RESEARCH STUDIES DONE IN DEAD BODIES WITH REFERENCE TO HBV AND OTHER BLOOD BORNE INFECTIONS

In one study of forensic autopsy cases, 32.6% had serological evidence of a significant viral infection and about 84%of known drug abusers were found to have at least one viral infection[14].

Zou et al., 2004.based on the investigations have shown that the rates of hepatitis B virus (HBV), human immunodeficiency virus (HIV), and hepatitis C virus (HCV) infections of tissue found higher in cadaveric donors than the first-time blood donors but lower than the normal general population

Yao et al. (2008) observed the musculoskeletal donors in Australia for viral infection and found that the incidence and prevalence of Human immune deficiency virus, Hepatitis B Virus and Syphilis among post-mortem bone donors was higher than that of the organ donors[33].

Yao et al., 2007showed similar results in his study of 12415 musculoskeletal tissue donors in Australia[34]. He also demonstrated the elevated incidence and prevalence of HIV, HCV and HBV infection in musculoskeletal donors than first time blood donors[33]

In live individuals if the donor was in the window period the result will come as false negative, but we can repeat the test by collecting the sample from the donor after a gap of 6 months and eliminate the previous

result and confirm the positivity but this chance of doing repeat test was not at all possible in our routine autopsies which undoubtedly leads to an increased risk in addition to the risk of testing the samples collected from the dead body in suboptimal fasion[33].

Weed and Baggenstoss, 1951; Brown et al., 1986; Roth et al., 1992; De Craemer, 1994; Healing et al., 1995; Kappel et al.,1996; Catteneo et al., 1999 stated in his study that the infectious pathogens which include Mycobacterium tuberculosis, hepatitis B and C viruses, HIV, and prions that cause transmissible spongiform encephalopathies in the cadavers present particular risks[35].

Barnett et al., 2001 stated in his study that specific serologic markers of hepatitis B and C viruses can be detected in cadaveric tissue banks (hepatitis B surface Ag 18.1% and hepatitis C Ab 14.3%[35]

Roth et al., 1992 stated the presence of specific serologic markers of hepatitis B and C viruses in postmortem blood tests for body donation programs[35]

Budka et al., 1995; Healing et al.,1995 stated the use of nonpermeable, disposable plastic sheet or similar material to avoid contamination of the dissection table[35].

Carolin et al conducted "A prospective time - course study on serological testing for human immunodeficiency virus, hepatitis B virus and hepatitis C virus with blood samples taken up to 48 h after death," as

such study do not exist at that time. The main objective of his prospective study was to investigate the post-mortem progression of serological parameters for HIV, HBV and HCV over time. The results of his study indicate a high post-mortem stability of antibodies and antigens. The low anti-HBc titres and probably the test- dependent discrepancies might be the possible reasons. EU Directive 2006/17/EC suggested that post-mortem blood samples should be collected within 24 hours. Till now only a few retrospective data about the post- mortem behavior of serological parameters of HBV, HCV and HIV parameters over time exist[36]. The post mortem samples should be centrifuged and stored at 2–6 ^{o}C without delay[34]. The post-mortem stability of these serological para- meters up to 48 hours increases the possible number of donors which can be used for transplantation.

Challine et al. (2006) correlated the post-mortem blood sampling time and macroscopic abnormal findings (especially haemolysis, and icteric or turbid sera) in his study on the post-mortem sampling interval (12, 12–24, 24– 36 and 36 hours) showed the significant increase in false-positive findings especially for HBsAg and anti-HIV and the increased retesting rate due to discrepant results in abnormal macroscopy and discarding of corneae. However, the post-mortem blood sampling time does not determine neither the prevalence of positive results nor their accuracy. Based on this study, the post-mortem blood sample timing has

no significant influence on the accuracy of the results. Due to the high number of false- positive results and the macroscopic serum changes authors recommended 12 h maximum time for drawing post-mortem blood samples.

Contrary to the findings of **Challine et al. (2006)** and **Heim et al. (1999)**, **Carolin Edleret al** noted minimal haemolysis when samples were collected after 36 h and moderate to severe haemolysis when samples were collected after 48 h of death but he neither found false-positive nor found false- negative results up to 48 h post-mortem in all his samples[34].

Li, Zhang, Constantine et al (1993)[37] studied and analyzed the risk factors connected with the Seroprevalence of HBV, HCV, HIV-1 in forensic autopsies, using ELISA. Serum samples were tested for HBV, HCV, HIV-1, HTLV-I and HTLV-II antibodies in 414 autopsy cases successively. Of the 414 cases, about 6% were positive for HIV-1, 23.2% for HBV, 19.1% for HCV, and 1.0% for HTLV-I and HTLV-II. In his study he established the increased HIV 1 prevalence when compared to general population at Maryland. The routine testing only for HIV-1 may likely miss other infections like hepatitis C virus or hepatitis B virus. Hence this study recommends the routine application of universal precautions for all autopsies.

Hossein Sanaei-Zadeh (2002)[38]studied the Seroprevalence of HBV, HCV and HIV in autopsies, at Tehran. Postmortem blood samples were collected in 173 cases for a period of 1 year, out of which 8 were positive for Hbs Ag and 7 cases were also positive for Hbs Ag and anti-HCV. No case was positive for anti-HIV 1 and HIV 2. He concluded that the medico legal autopsy cases which we thought to be at low risk are not so and must be considered as both highly and dangerously infectious. Hence universal precaution must be carried out during autopsies.

Eriksen MB et al (2009)[39]collected the autopsy blood samples from the drug addicts in Denmark and screened them by polymerase chain reaction (PCR) technique for HIV antibodies and hepatitis antibodies with rapid kits and reported the Postmortem Detection of HIV genome in about 40% of samples and Hepatitis B virus genome in about 20% of anti-HBc – positive/anti-HBs-negative samples.

Weston, J. and Locker et tal[40] in his study after assessing 44 postmortem of adults noticed a significant number (8.3%) of glove punctures, out of which 31.8% went unnoticed. Although we know that gloves are impermeable to HIV and HBV infection, punctured gloves are not so. The results of this study showed that evisceration technique which is the first procedure in our routine autopsy techniques is an especially risky procedure in terms of glove puncture. As the personnel with preexisting hand lesion may be bathed in the infected material for several

hours due to the rent in gloves and was never treated according to current safety measures. The risk of contamination from the puncture associated with concurrent injury is lesser than puncture without concurrent injury, as it may be noticed immediately and attended promptly as per the universal guidelines. The habit of frequent change of gloves and hand washing throughout the procedure and also at the end of procedure is the most important way of reducing the infection associated with glove puncture

Li et al[37] and **Plessis et al**[41] in their study in forensic autopsy performers reported the prevalence of hepatitis B and HIV as 23 and 8% respectively. The purpose of their study is to determine whether autopsies of the corpses which have been presumed so far to be of low risk group are safe or not. HBV has more transmissibility rate as blood borne and as aerosol than HIV. As there was limited data available regarding these risks to forensic medical personnel who are exposed daily to large numbers of severely traumatized bodies in Iran they conducted a research to identify the seroprevalence of these viruses in a low risk forensic autopsy population in Tehran, the capital of Iran[38]

Healing et al., 1995 in his study stated that the cadaver handling workers are exposed to a number of infectious agents[42].

Rischetelli et al, 2001 in his study stated that most likely infections are those produced by blood born viruses, enteric pathogens and mycobacterium tuberculosis[42].

Curti and Biran, 2001 in his study stated that a strong aversion to dead may represent a natural instinct to protect our self against the disease[42].

De .ville, 1980 in his study stated that the microorganisms involved in the decay process are not always pathogenic[42].

Morgan, 2004 stated that the belief that the dead bodies are infectious can be considered as natural reactions by the persons to protect themselves from disease[42].

Kermode et al. examined the occupational exposure to blood and the risk of blood-borne virus infection among health-care workers in rural India and found that 63% of workers reported at least one percutaneous injury in one year when compared to 24% in the US population[43, 44]

A study "Risk of infection among primary health workers in the Western Development Region, Nepal" suggests that basic health workers lack sufficient knowledge of universal precautions. The majority of respondents (59%) did not answer universal precaution knowledge questions. Only 22% of the workers reported accurate knowledge of universal precautions as an effective barrier between health workers and patients to prevent the transmission of infections[45].

HISTORY

The earliest record of an epidemic caused by hepatitis B virus was made by Lurman in 1885 [46]. In 1883 an outbreak of smallpox occurred in Bremen and 1,289 shipyard employees were vaccinated with lymph from other people. Eight months later, 191 of the vaccinated shipyard employees became ill with jaundice and were diagnosed as suffering from serum hepatitis. Those employees who had been inoculated with different batches of lymph remained healthy.

Lurman's paper which proved that contaminated lymph was the source of the outbreak was now regarded as a classical example of an epidemiological study. Later in 1909, numerous similar outbreaks were reported following the introduction of hypodermic needles that were used and more importantly reused for administering Salvarsan for the treatment of syphilis. Until 1966 the virus was not discovered when Baruch Blumberg who was working at the National Institutes of Health (NIH) discovered the Australia antigen (later known to be hepatitis B surface antigen or HBsAg) in the blood of Australian aboriginal people[47]. Since 1974, the year in which the research was published by MacCallum, the role of virus had been suspected [48] but in 1970 D.S. Dane and others discovered the virus particle by electron microscopy.[49] By the early 1980s the viral genome had been sequence[50] and the first vaccines were being tested[51].

INCIDENCE AND EPIDEMIOLOGY

All over the world more than 33% of the population has been estimated to have HBV infection with highest incidence of HBV seropositivity (anti-HBsAg) noted in sub-Saharan Africa, the Far-East. More than 8% of the populations are found to be infected in northern Canada, northern South America, Greenland and Alaska with a lifetime risk of infection of more than 60% with infections in childhood.

About 5% of the populations are chronic carriers of HBV and nearly 25% of all carriers develop serious liver diseases such as chronic hepatitis, cirrhosis, and primary HCC. Hepatitis B Virus infection causes more than one million deaths every year.[24, 52, 53, 54]

De Ville, 2000; **De Ville de Goyet C 2000** stated that the risk of infection in case of hepatitis B depends on infectious status of victim, mode of exposure and vaccination status of the exposed individual. In many developing countries, prevalence of chronic hepatitis B is around 8% to 10% **(**Stop Propagating disaster myths. Lancet, 356:762-64) and was high for the pathologists, surgeons and other medical personnel exposed to blood.[34, 42]

The incidence of the HBsAg carrier state in populations is related most importantly to the incidence and age of primary infection[52]. There is a variation ranging from 0.1 to 20% in the HBsAg carrier rate among different populations groups all over the world.

In areas of low seropositivity where anti-HBsAg antibodies is seen in less than 2% of the population , the lifetime risk of HBV infection is less than 20% with most infections occurring in adults who are in elevated risk groups.

The highest incidence of the disease is seen among the teenagers and young adults in low-risk areas of the world. General population had a lower incidence of Hepatitis B Virus infection when compared to certain high risk groups which have a higher incidence. Nevertheless, screening techniques used in blood donors, improved sterilization procedures used for blood derivatives and the availability of newer and effective vaccine have lowered substantially the infection risk.[24]

In less than 2% of the US general population, HBsAg seropositivity is noted, where as 10-15% of the Asian Americans have chronic HBV. In America, 50 % of the children were born to Asian Americans mothers with chronic HBV infection. HCC is a leading cause of death in Asian Americans. There is no difference in the seropositivity between the Asian American and African Americans as both have a higher rate.

According to the CDC, approximately 78,000 people in the US were infected by Hepatitis B Virus in 2001 and about 5,000 people die per year from Hepatitis B Virus-associated disease. In United States, 1 in 20 people is infected by HBV at some time in their lives with the highest

infection rate being in young adults. About 5% of people infected by HBV get a chronic infection and there are more than one million Americans with chronic hepatitis B. Up to 25% of chronic HBV infected patients will die due to some form of liver disease. In United States the acute hepatitis B infections has decreased considerably due to the excellent vaccine which are now currently available

ENDEMICITY

Primary HBV infections have no seasonal preferences.[24, 53]

Hepatitis B is highly endemic in all areas of Africa, Alaska, Northern Canada, some parts of South America and parts of Greenland, the Eastern Mediterranean area, Eastern Europe, China, South-East Asia, and the Pacific Islands, except Japan, Australia and New Zealand with 5 to 15% chronically infected HBV carriers in most of the above said areas. Co existence with HDV is also noticed in some areas where the liver of infected patients has got severe damage.[52, 55]

The countries such as US has low endemicity and higher mortality rate for HBV when compared to Haemophilus influenza b (Hib)(five times) and measles (ten times) before the introduction of routine vaccination of children.

The epidemiological patterns in endemic areas of Asia and Africa are different from those seen in Western Europe and North America. Transmission from the mother to new born and from to child through

close contact is cause for most infections which occur in infants and children in these regions. Transmission of virus through skin contact with contaminated needles or not following safe injection techniques is also a possibility in the above mentioned countries.[24, 52]

HBV TRENDS

HBV has no seasonal trends[24].

The use of contaminated blood or blood products or unsterile injections leads to the epidemic spread of HBV infection.

Most HBV infection in young children is not detected in surveillance studies of acute disease as they are asymptomatic. So the vaccination coverage data and population-based serological analyses are used to assess the effectiveness of vaccination programs implemented in infants. The success of the programme and decreased rate of infection is indicated by a fall in the prevalence rate of chronic disease [52]

The prevalence of chronic HBV infection has considerably reduced even in high endemic areas because of the successful implementation of infant immunization programmes.[52]

The routine infant immunization which when successfully implemented will prevent HBV transmission among all age groups by inducing broad population based immunity to HBV infection. But only in longer term it will have an effect on the incidence and severe consequences of chronic HBV infections[56].

PREVALENCE

HBV occurs worldwide.[52, 57]

The highest rates of HBsAg carrier rates are found in developing countries with primitive or limited medical facilities.[52]

In areas of Africa and Asia, widespread HBV infection may occur in infancy and childhood and the overall HBsAg carrier rates may be 10 to 15%.

The prevalence is lowest in countries with the highest standards of living, such as United States, Great Britain, Canada, Scandinavia, and some other European Nations.

In North America, HBV infection is most common in young adults. In USA and Canada, serological evidence of previous HBV infection varies depending on age and socioeconomic class. Overall, 5% of the adult USA population has anti-HBc, and 0.5% is HBsAg positive.

In developed countries, HBV exposure may be common in certain high-risk groups.

Adult population infected with HBV usually acquires acute hepatitis B and recover, but 5 to 10% develop the chronic carrier state. Children infected with HBV rarely develop acute disease, but 25 to 90% become chronic carriers. About 25% of carriers will die from cirrhosis or primary liver cancer as adults.[52, 58]

In the past, there were high risk for HBV infection to the recipients of blood and blood products. Over the last 25 years it was made mandatory to test the donor blood for the presence of HBsAg which had improved the sensitivity to greater extent.

In 19% of countries routine testing of all blood donations for HBsAg was not done according to an unpublished data. The routine and systematic screening of blood collected from donors for the presence of HBsAg before transfusion reduces the residual risk of HBV transmission to a minimum in many countries. The additional viral inactivation and removal procedures used for plasma derived medicinal products (including antihaemophilic factors) leads to reduction or no HBV transmission by these products.

Inspite of all the necessary precautionary measures taken, the developing countries still have the risk of HBV transmission. The contaminated needles and syringes used in clinics or by acupuncturists and tattoo parlor may lead to an outbreak of hepatitis B. HBV transmission from a HBsAg positive health care workers has also been documented rarely.[59]

The reduction in Age-related prevalence of HBsAg even in highly endemic countries and the adoption of universal immunization of infants suggested the possibility of HBV eradication from humans.

The use of Hepatitis B vaccines by hundreds of millions of people from the year it was available (1982) had and an impact on the disease and reduced the prevalence rate of HBV carriers from high to low in many countries.

Fig 1: Geographical Distribution of Prevalence of Hepatitis B Infection in 2005

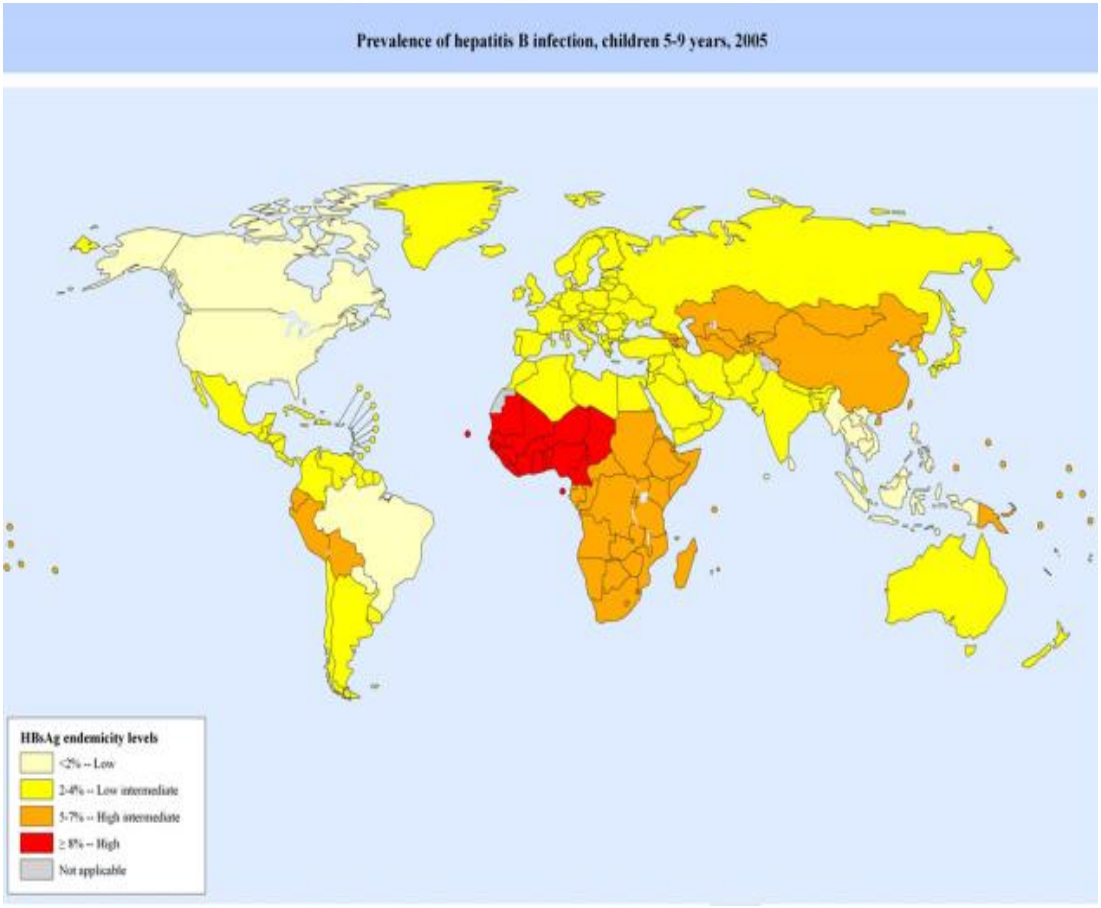
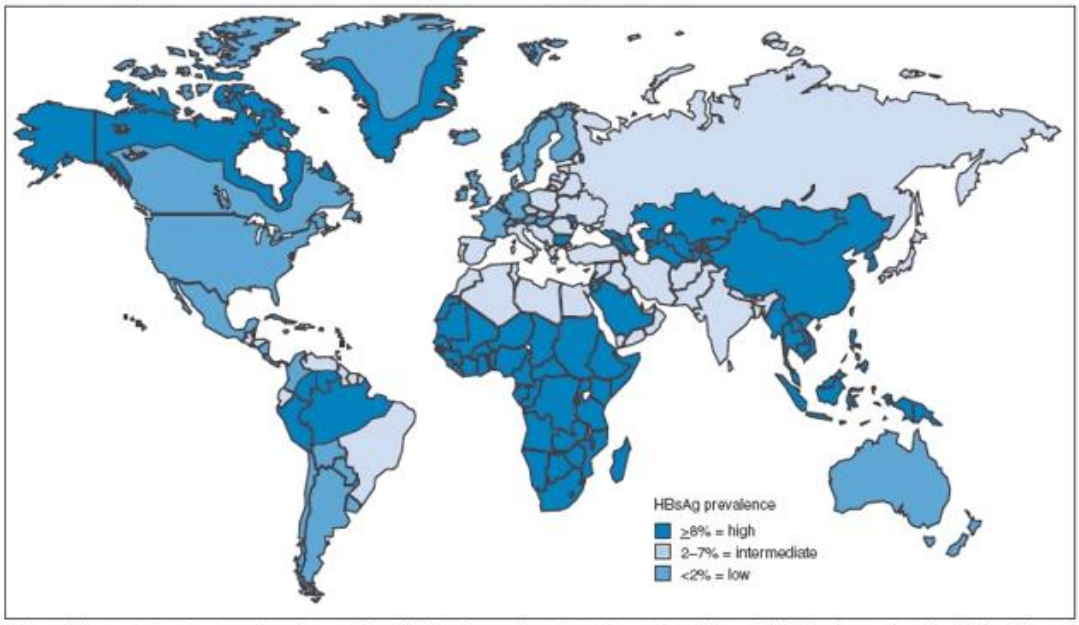


FIG 2: Geographical Distribution of Chronic Hepatitis B Virus Infection in 2005

FIGURE 1. Geographic distribution of chronic hepatitis B virus (HBV) infection, 2005*



* For multiple countries, estimates of prevalence of hepatitis B surface antigen (HBsAg), a marker of chronic HBV infection, are based on limited data and might not reflect current prevalence in countries that have implemented routine childhood hepatitis B vaccination. In addition, HBsAg prevalence rates might vary within countries by subpopulation and locality.

Fig 3: Geographical Distribution of Hepatitis B in 2007

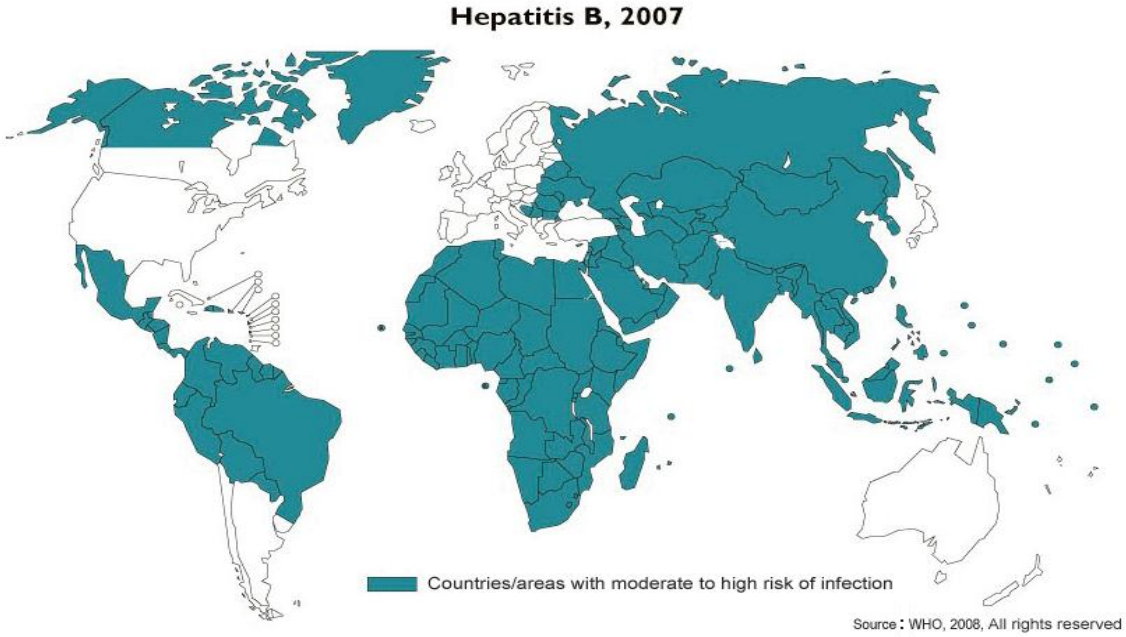
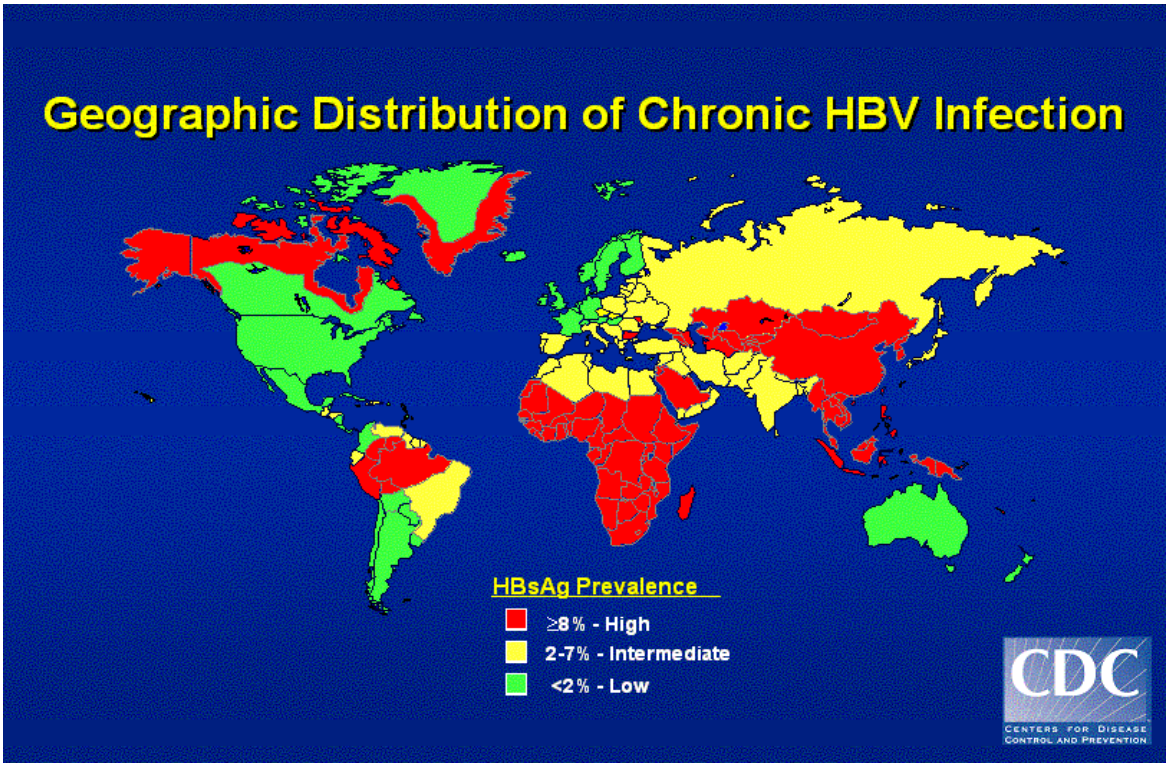


Fig 4: Geographical Distribution of Prevalence of Hepatitis B Surface Antigen

Fig 5: Geographical Distribution of Chronic HBV Infection



HEPATITIS B VIRUS

Hepatitis B virus (HBV) is a partially double-stranded circular DNA virus belongs to the hepadnavirus family and has a DNA genome that is replicated via an RNA intermediate. The virus consists of an envelope containing the surface antigen (HBsAg) which surrounds the viral DNA containing core capsid. During replication of HBV, both the whole intact virions and incomplete virus particles consisting entirely of HBsAg are produced.

Fig 6: Structure of HBV

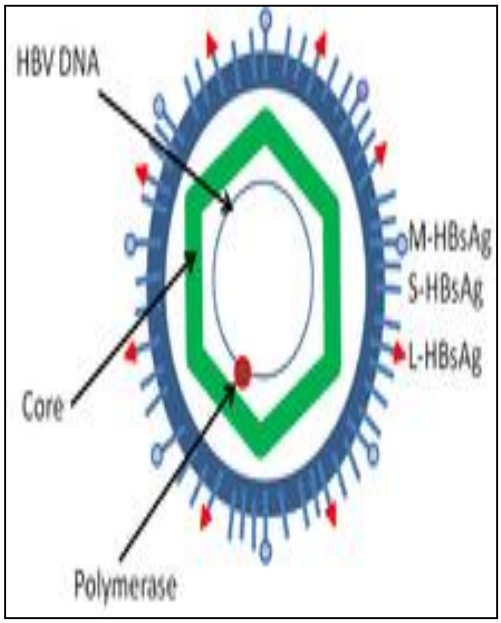


Fig 7: Electron Microscopy - HBV

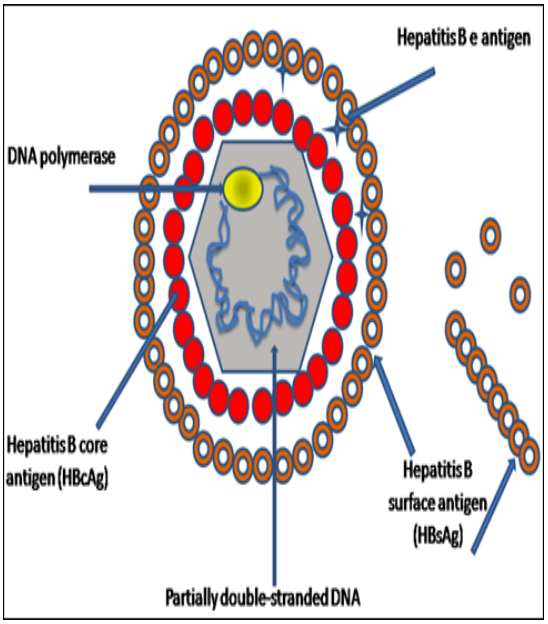


Fig 8: Hepatitis B surface antigen or HBsAg

The envelope of Hepatitis B Virus consist of most important protein named as “Hepatitis B surface antigen” or “HBsAg”, which was called as Australia antigen in older days. This antigen contains the determinant "a", which was common to all known viral subtypes and which is immunologically distinguished in two distinct subgroups (ay and ad). HBV has 10 major serotypes and four HBsAg subtypes have been recognized (adw, ady, ayw, and ayr).

Components

It consists of:

- HBsAg
- HBcAg (HBeAg is a splice variant)
- Hepatitis B virus DNA polymerase

- HBx. The function of this protein is not yet well known[60] but evidence suggests it plays a part in the activation of the viral transcription process[61]

SEROTYPES AND GENOTYPES

The virus is divided into four major serotypes (adr, adw, ayr, ayw) based on antigenic epitopes presented on its envelope proteins, and into eight genotypes (A-H) according to overall nucleotide sequence variation of the genome. These genotypes have a distinct geographical distribution and are used in tracing the evolution and transmission of the HBV. Differences between genotypes affect the course and severity of the disease, likelihood of developing complications and response to treatment and possibly HBV vaccination [62, 63]

Genotypes differ by at least 8% of their sequence and were first reported in 1988 when six genotypes were initially described (A-F) [64.] Two further types (G and H) have since been described.[65] Most genotypes are now divided into sub genotypes with distinct properties.[66]

PATHOLOGY

As soon as the HBV enters the bloodstream it targets the hepatocytes where its receptor found predominantly. The initial dose of viral load entering the body decides the rate at which symptoms appear.

Even though the viral replication will start only after a few days of initial infection, the incubation period ranges from 45-180 days.

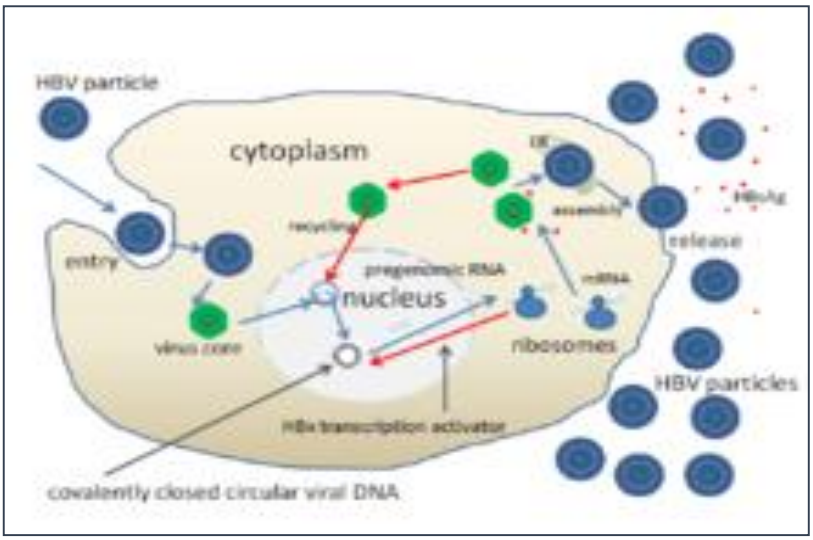
Ground glass appearance in infected cells, the characteristic appearance of HBsAg was the first sign of HBV infection. Like other hepatitis viruses, Immune-mediated symptoms and also the resolution of disease were produced by inflammatory and cell-mediated responses which are targeted against the HBsAg which lies on the surface of hepatocytes.

Chronic infection produced by HBV increases the risk of morbidity and mortality of the individual from chronic liver disease or primary HCC. The initial event which cause the virus to enter the body and the onset of symptoms which constitutes loss of appetite, nausea, vomiting, abdominal pain, fatigue, jaundice and joint pain was often masked by the long incubation period of 6 to 24 weeks[67]

Younger the patients, lesser will be the cell-mediated immunity and milder the symptoms. Within five years, as a result of lesser immune response to HBV, chronic infection develops which subsequently lead to chronic hepatitis which in turn leads to development of cirrhosis in up to a quarter of patients. Up to 25% of these patients will develop liver failure or HCC which are fatal in the absence of a liver transplant.

In age group of less than five years about 10% of patients show clinical illness (jaundice). In young age group 30-90% of patients infected with HBV develop chronic hepatitis. In age group of 5 years and above, though 30-50% of patients have symptoms, 30% have no clinical symptoms and 2 to 10% will develop chronic hepatitis.

Fig 9: HEPATITIS B VIRUS REPLICATION



PATHOGENESIS

After certain period of time which is usually around 30 to 50 years, persons who contracted this infection early in their life may develop chronic hepatitis, which is usually followed by cirrhosis, and finally HCC. Regarding the infection, males when get the infection they usually have this infection for long period of time when compared to females. Antibodies will develop soon after the infection in case of females.

The main reason for the carcinogenic effect of virus is not the direct effect of virus, till now oncogene related to the virus or any mutagen is not demonstrated. The possible reason for the occurrence of carcinoma in the infected patient is due to the chronic liver disease, after the infection there will be inflammatory reactions along with some amount of liver damage, when these damages progresses further to cirrhosis then the chance for the development of malignancy in that particular infected individual is very high[53].

Thus hepatitis B infection alone does not cause the carcinoma, for the occurrence of carcinoma the liver damage should accompany definitely. This can be proved by, in many healthy persons who are carrier for antigen of hepatitis B does not developed carcinoma further throughout the life period of time.[68]

The liver cells in persons affected with this infection are get injured by three various mechanism.

- When the person infected with hepatitis, our immune system will generate cytotoxic T cell, which will act on the various antigens which is present on the virus, this will produce a injury on the hepatocytes.[52,53,57]

- The antigen within the virus also acts on the liver cells directly mainly the core antigen which is considered as other possible mechanism.[52,53,57]
- Sometimes the antibodies are not secreted in proper quantity, which also contributes for the injury of the cell.[57]

POST MORTEM STABILITY OF HBV

Brendan Jacka et al after analyzing the data from his study demonstrated the stability of immunoglobulins and albumin in specimens collected up to 24 hours port-mortem[69].

Though the concentrations of total protein and albumin were more likely to be outside the normal reference range than random living controls, there was no correlation between the age of the donor and the concentration of any of the biochemical markers. This study also supports the stability of serological markers of infection and indicators of plasma dilution in cadaveric specimens[69].

In cadaveric specimens, the plasma dilution which may occur as a result of autolysis and degradation has been determined to be of little consequence and another consideration is the interactions of degraded blood components with the serological screening assays employed to identify infection. Potential inhibitory effects caused by abnormal concentrations of biological substances have been identified in living

donors by the manufacturers of serological screening assays - for example haemoglobin and bilirubin[69].

As cadaveric specimens may exhibit different levels of biological substances, it is necessary to determine if these changes also influence the result of serological screening assays. This study is the first reported assessment of the test kits, where the clinical and analytical sensitivities were found to be equivalent to results from random living donors. While these assays were able to detect the presence of serological markers of HIV, HCV and HBV infection accurately and reproducibly, there was a greater rate of reactivity in initial testing than seen for the general population of Sydney[69].

Subsequent confirmatory testing was required to determine whether the instances of reactivity were the result of assay interference or actual infection. High levels of false-reactivity which have been reported when using cadaveric specimens in serological screening, suggests that the high rate of initial reactive results seen in this study, are a function of the specimen quality[69].

The present study had demonstrated that plasma dilution is not occurring in cadaveric specimens collected within24 hours from death, and that concentrations of immunoglobulins are relatively stable when compared to living donors.

Subsequently, accurate and reproducible detection of serological markers of infection should be expected when using these assays with cadaveric specimens. Accurate reporting of reactive specimens using these assays must be further validated using repeat testing and confirmatory assays. In conclusion, the safety of transplant recipients should be maintained to the current standards of living donors when using post-mortem specimens for testing[69]

Heim et al. 1999; Miedouge et al. 2002 stated that there will be a increased incidence of false seropositivity in post mortem blood samples tested for serological markers of viral infection, as the composition of blood will change due to potential changes which occur in the vascular compartments after death which makes the screening methods used for sera from cadaveric organ donors need further validation before testing these[33].

Turner et al. 1997 stated that the increased rate of organ rejection and reduction in availability of organs for transplantation is due to false-positivity which are observed in the results of any marker of disease [33]

Singer et al. 2008 stated that he concerned more about the false-negative results which is to given more importance and eliminated, as it creates a false belief about the safety of the donor organ and thus reduces the safety for recipients[33].

Challine et al. 2006 after observing the previous studies he told that the immunoassays and NAT assays are inhibited by the presence of free Hb in post mortem serum samples [33]

The factors which potentially compromise the sensitivity of post mortem serological screening tests:

(a) Post mortem dilution of the serum in the vascular system affecting the detection limits

(b) Post mortem haemolysis which leads to the release of free Hb which in turn inhibits the interaction between Ag-Ab complex and reduces the sensitivity and causes false seronegativity

(c) Post mortem proteolysis and its interference with Ag-Ab binding and cause false-positivity.

So their aim was to define the correlation between the post-mortem serum changes and its influence on tests done to detect infection with HIV, HCV, HBV, T. Pallidum and HTLV I/II in donors by comparing the pre and post-mortem samples analytical performance[33].

Heim et al. 1999 stated about the post mortem biochemical changes in blood specimens and the interference of increased free serum haemoglobin with the immunoassays which are often used as a serological screening modality for infectious disease[33]

Heck and Baxter in 1994 and Eastlund in 2000 stated that the haemodilution caused by the transfusion of blood and infusion of

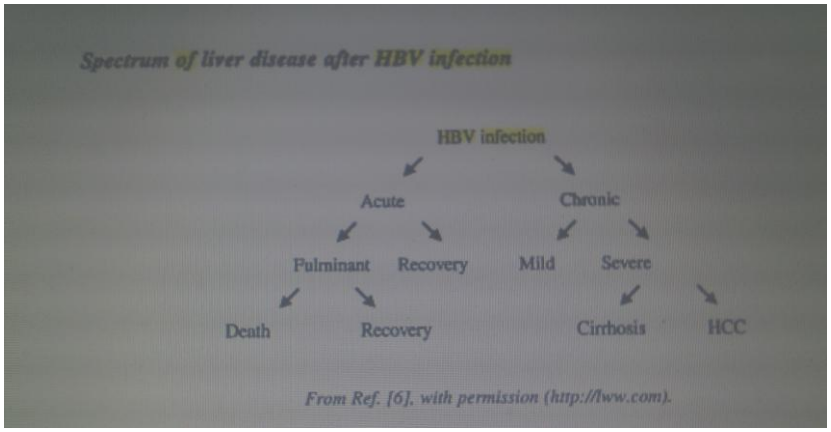
crystalloids in an effort to save the life during the last hours of life, also affects the serological tests done for screening the infectious diseases[33]

Eastlund and Schuller1994 contradicts the statement of Heck and Baxter and demonstrated that sensitivity of these assays would not be affected by resuscitation induced haemodilution as negative results occur only when the level of haemodilution is very high which is not normally seen in resuscitation[33]

This study showed that there was no significant difference between antibody and antigen markers in pre mortem and post mortem specimens. The results obtained from testing the biochemical parameters in post mortem blood suggest that the levels of immunoglobulinlevels (total or specific) in the post mortem samples will not be reduced due to the post-mortem stability of antibodies[33]

Kitchen and Newham in 2010 did the first study to analyze the sensitivity of the Abbott Architect Analyser by mixing the known positive sera with cadaveric sera to make the sample similar to post-mortem samples. They have also assessed the post mortem changes and established the post mortem stability of infectious viral seromarkers up to 24 h post mortem and made the results more reliabile[33].

Fig 10: Spectrum of liver disease after HBV infection



The factors correlating the severity of disease are the infecting viral dose and the age of the person infected with HBV[52, 57]. 90% of adult population and 10% of new born had transient HBV infection, whereas the rest had the persistent infection with HBV but clinically detectable only in smaller groups[52]. Clinical manifestation of jaundice was observed in infected children (<10%) and adults (30-50%)[70]

Primary infection had a wide range of clinical manifestation starting from mild to severe to fulminant with or without liver damage[57]. Majority of the patients having acute infection had sub clinical presentation with progression to fulminant damage in less than 1% of symptomatic[57] ·All over the world, Chronic infection of the patients with HBV is seen in 350 million people[54] .Even though, 33 % of the patients with persistent HBV infection had associated cirrhosis and HCC, liver function and histology was found to be normal in some cases[57.]

VIRAL MARKERS OF HBV SEEN IN SERUM

The nature of the viral markers present in the serum of infected patients varies with the duration of infection (Acute or Chronic).[52, 57, 71]

Table 1 : Serological Viral Markers

ANTIGENS	ANTIBODIES
HBsAg Earliest indicator of acute infection and chronic infection. Useful in diagnosis and screening	anti-HBs Appear 30 to 120days after appearance of symptoms Presence indicates clinical recovery and development of immunity.
HBcAg Not detectable in the bloodstream. Marker of the infectious viral material Most accurate index of viral replication.	anti-HBc Identifies all persons with previous infections and HBVcarriers, but it cannot differentiate carriers from non-carriers
HBeAg Its presence indicates that the patient is infectious and if persistent beyond 10 weeks shows infectiousness and progression to chronic infection.	anti-HBe Its presence is prognostic for resolution of HBV infection. If HBsAg, anti-HBs and core HBV mutants are absent in the given sample with presence of anti-HBe along with anti-HBc, it indicates that the patient is of low contagious in nature and in convalescence phase[57.]

HBxAg Detected in HBeAg positive blood in patients with both acute and chronic hepatitis	anti-HBx When other virological markers are becoming undetectable, it appears.
HBV DNA Detected as soon as 1 week after the initial infection and used in monitoring of the treatment with antiviral drugs and to detect mutants which the currently available methods failed to detect.	
HBV DNA polymerase Though detected early (within 1 week of initial infection) has right now no role except for research.	

PATTERNS OF VIRAL INFECTION

All patients exposed to HBV will not react in the same way and hence had varied patterns of infection.[57]

1. HBsAg-positive Self-limited primary HBV infection
2. HBsAg-negative Self-limited primary HBV infection
3. HBsAg-positive persistent HBV infection

Unusual HBV serological profiles which require further evaluation

The sample which shows unusual serological profile should be retested or new sample to be collected and tested[24.]

Table 2: Unusual Serological Profile and its Interpretation

Serological Profile	Interpretation
HBsAg positive and anti-HBc negative	Acute hepatitis B patients in incubation period (even before the onset of clinical symptoms and biochemical derangements).
Positive for all the three **HBsAg** **anti-HBs** **anti-HBc**	Seen in acute hepatitis B resolution, in chronic carriers with serious liver disease or in carriers exposed to heterologous subtypes of HBsAg.
Only anti-HBc positive	Infection in the past which was not resolved completely
HBeAg positive and HBsAg negative	Not commonly seen
Positive for both **HBeAg and anti-HBe**	Not commonly seen
only anti-HBs positive (in a non immunized person)	Those who received 1. passive transfer of anti-HBs following blood transfusion from a vaccinated donor 2. clotting factors 3. immunoglobulins In new born children born to HBV infected mothers irrespective of whether the infection is recent or past. Seen in forgotten immunization status!

Clinical phases of acute hepatitis B viral infection

Acute infection with Hepatitis B virus infection comprises > 90% of adult-onset infection. The remaining population of adult-onset infection and >90% of new born infection turns to chronic and continue for the rest of the life with small percentage of mortality from acute HBV.

Patients with acute illness often have an insidious onset with non specific symptoms. Jaundice may develop later with or without Pyrexia.[24, 52, 57, 68]

The initial non specific symptoms is followed by the icteric phase which manifests through dark appearing urine, pallor of stools and yellowish staining of body tissues and fluids within 10 days. Jaundice needs the level of total bilirubin in blood to be in excess of 20 to 40 mg/l to become clinically apparent. It is accompanied by the enlargement of liver and spleen. The disease resolves with the disappearance of jaundice and the appearance of antibodies (anti-HBs) after 4-12 weeks of initial infection in about 95% of adults[24.]

After 4-8 week of acute illness, the disease often resolves spontaneously in most patients without any adverse sequel and recurrence. Morbidity and mortality is high in elderly persons due to the development of fulminating and fatal acute hepatic necrosis. Young

children has the tendency to become chronic carriers if they acquire the infection before the age of seven and acute illness will develop rarely in them.[24, 52, 53, 57, 68]

Serum shows the presence of anti-HBc IgM serum antibodies during acute phase and the presence of anti-HBc IgG serum antibodies during convalescence and recovery. During the transient period (<6 months) HBsAg, HBeAg, and viral DNA will be present. During seroconversion, viral markers which were present already in the circulation will be cleared with the appearance of anti-HBsAg and anti-HBeAg.

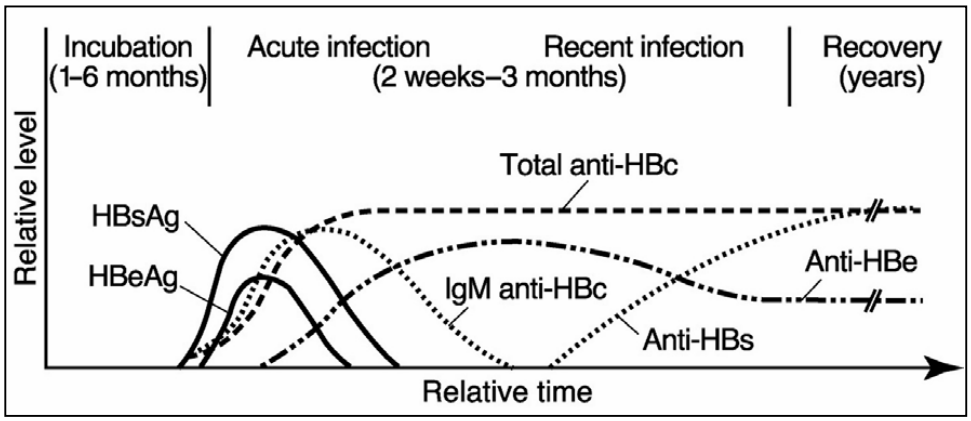
Though the increase (Mild to Moderate to Severe) in serum transaminase levels (ALT) during acute phase is the hallmark of acute viral hepatitis, a striking increase does not reflect a poor prognosis.

The amount of viral load in the inoculum, the mode of virus transmission into the host and host factors determines the variability of the incubation period from 45 to 120 days (average of 60 to 90 days)[24, 52, 57, 68]

If the large amount of virus enters into the circulation, patients develop icteric symptoms early due to the shorter incubation period, which is common in transfusion of blood which is already infectious[57.]

No dietary or life style modification is needed in most cases.

Fig 11: Serological Pattern In Acute Viral Infection



CLINICAL FEATURES OF CHRONIC HEPATITIS B VIRUS INFECTION

- Persistence (>6 months) of HBsAg (with or without concurrent HBeAg) is the principal marker for the risk of developing chronic liver disease and hepatocellullar carcinoma (HCC) later in life.
- The presence of HBeAg indicates the highly contagious nature of the blood and body fluids of the infected individual

Low replicative phase:

This phase is associated with the loss of HBeAg or a decrease or loss of the HBV DNA concentrations and with the appearance of anti-HBe. A decrease in inflammatory activity is evident histologically.

Serologic changes like the loss of HBV DNA and HBeAg are referred to as seroconversion.

Non replicative phase:

Markers of viral replication are either absent or below detection level and the inflammation is diminished but if cirrhosis has already developed, it may persist indefinitely.

The laboratory abnormalities consist of elevation of the ALT which ranges from normal to 200 IU/l in up to 90% of patients. Transaminases, serum bilirubin, albumin, and gamma globulin values are mild to markedly elevated. Autoimmune antibodies such as anti-nuclear antibody, anti-mitochondrialantibodyand anti-smooth muscle antibodymay be present[24.]

Sustained increases in the concentrations of the aminotransferases together with the presence of HBsAg for >6 months is regarded as indicative of chronic hepatitis.

Progression to fulminant hepatitis B

Fulminant hepatitis B is a rare but fatal condition caused by massive hepatic necrosis which develops in about 1% of cases.[24, 52]

The children had a better prognosis when compared to adults, in whom the survival is uncommon and if survived, only a few persons have a complete recovery. Permanent damage to the liver and chronicity of infection was not noted in the survivors[24, 57.]

Those infected with precore mutants often develops severe infection and early damage to the liver, in form of chronic hepatitis and development of early fulminant hepatic failure[72.]

There is an association of genetic heterogeneity of HBV, immunological factors of the host, coinfection or super infection with other viral hepatitis agents with the development of fulminant hepatitis B[24, 57.]

In patients with fulminant hepatic failure, as the hepatocytes are being lost the rapid fall in ALT and AST should not be interpreted as a resolving hepatic infection and the outcome is often fatal[71.]

Fig 12: Serological Pattern in Chronic HBV infection (HBeAg positive)

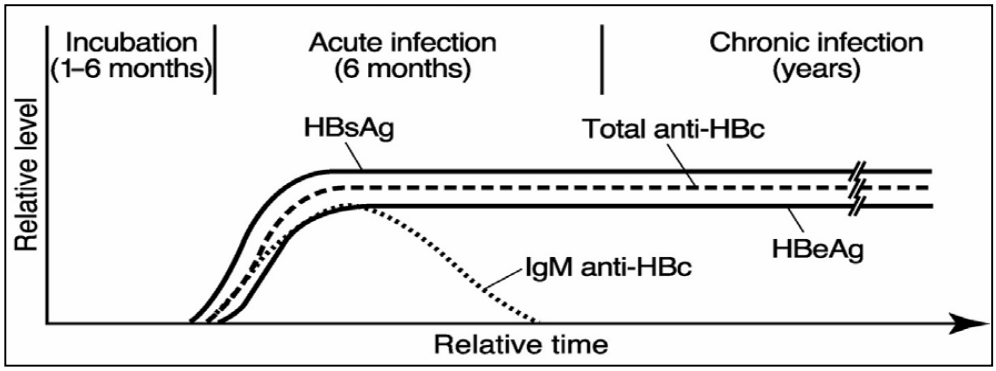
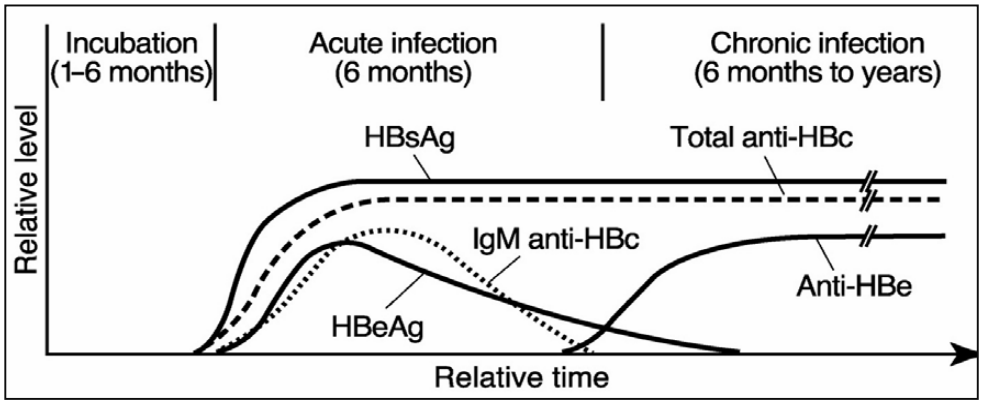
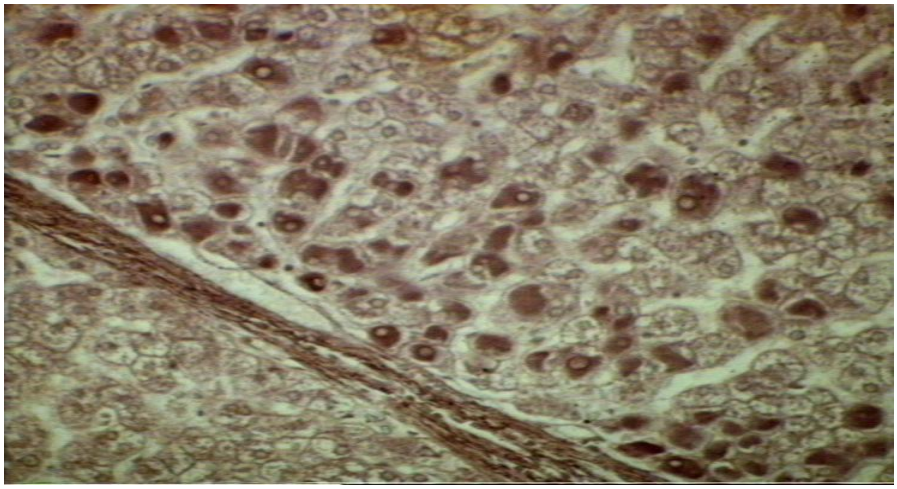


Fig 13: Serological Pattern in Chronic HBV infection (HBeAg negative)



HBV and Cirrhosis

Fig 14: HPE of Liver in Cirrhosis



Chronic hepatitis B viral infections were one among the 10 leading causes of mortality in men. It is estimated that men has 40 – 50% lifetime mortality risk from cirrhosis and/or hepatocellular carcinoma which develops as a sequel of chronic disease. Women pose lesser risk (15%) when compared to men.

The liver substance undergoes chronic and wide spread destruction leading to development of cirrhosis in up to 20% of the patients with chronic persistent hepatitis. The internal structure of the liver in cirrhotic patients is grossly deranged in such a way to obstruct the blood flow, which cause the liver cells to die with decrease in liver function. The liver parenchyma is damaged by recurrent HBV stimulated immune responses and was progressively replaced by fibrotic tissue which leads to the formation of nodule (micro or macro). As the liver inflammation is asymptomatic, patient will be unaware of the inflammatory progression and subsequent development of cirrhosis. The presence of HBV DNA determines the contagious nature of most of the carriers.

HBV and hepatocellular carcinoma (HCC)

Over Worldwide, it is most common in both the males and females with seventh place in the order of frequency in males and ninth place in females. There is sexual, racial, geographical and chronological variation in the incidence of HCC. In fact, HBV infection may be the cause of over 80% of primary hepatocellular carcinoma cases worldwide. This is particularly the case when the patient is HBeAg-positive.

HCC is one among the three most common causes of deaths in males due to carcinoma in East and South-east Asia[57]

HCC has a higher mortality with more than 500 000 deaths annually with male preponderance (M : F = 4:1) throughout the world. In a given general geographic area, the HCC and persistent HBV infection follows the same frequency in distribution pattern. After analyzing the age at which the tumour appears after HBV infection in patients with clinically recognized tumours, it was suggested that tumour appears 35 years after the infection with HBV[24, 57.]

Though a number of patients with chronic hepatitis will develop hepatocellular carcinoma[24, 57], those who acquired the infection in early childhood[52] are at an increased risk. Though only 5% of cirrhotic patients develop HCC, Cirrhosis was noticed in 60 to 90% of HCC patients.[24, 53, 57]

In Taiwan where 15% of the population are HBsAg carriers who had a 217 times risk of developing hepatocellular carcinoma than that of a non-carrier. About 50% of deaths of HBsAg carriers are caused by liver cirrhosis or hepatocellular carcinoma when compared to 2% of general population.

The 25 to 60% mean 5-year survival rate which was observed in patients whose hepatocytes undergo malignant transformation, which leads to the development of HCC, depends on various parameters such as the symptoms, tumour size and its respectability and the presence or

absence of α-fetoprotein (AFP). AFP- positive tumours which was not resectable have a mean survival rate of 5 months, whereas AFP-negative tumours have 10.5 months[24.]

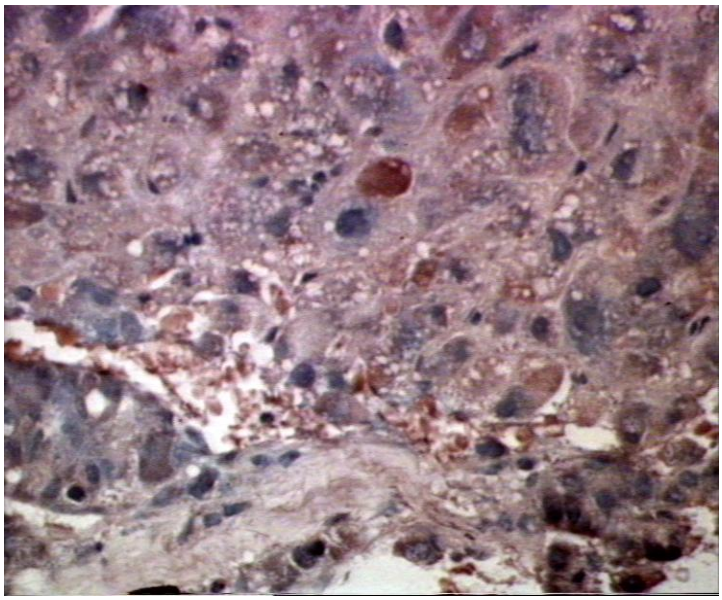
HCC can often be detected at a resectable stage in HBsAg carriers by liver scanning or ultrasound procedures, when during the serial follow up the serum AFP rises significantly above the patient's own baseline (>100 μg/ml)[57]. This suggests that HBsAg carriers (those above 40 years and those with cirrhosis) should have regular (at 6 months intervals) and serial serum AFP determinations and ultrasound examinations[57.]

Patient often will die if HCC presents clinically, with survival period of less than 3 months whereas, if detected early, there are various treatment modalities available such as surgery, radiotherapy, and chemotherapy to give an 85% cure rate

Sundaram C et. Al did his study on 206 cases of autopsy livers for the presence of hepatitis B surface antigen by orcein staining to find the association of hepatocellular carcinoma and cirrhosis with hepatitis B surface antigen in Andhra Pradesh State, in South India. Of 114 cases of cirrhosis, 67.54% were positive for the antigen. The antigen positivity was 100% in macronodular (13 cases), 98.7% in mixed (55 cases) and 21.74% in micronodular cirrhosis (46 cases). Only 50 out of 58 cases

of HCC were associated with cirrhosis. Hepatitis B surface antigen was demonstrated in 80% of HCC associated with cirrhosis and in 75% of cases of HCC not associated with cirrhosis[73].

Fig 15 : Hpe of Liver in HCC



Hepatitis B - Extra hepatic manifestations

Seen in 10-20% of patients as

1. Transient serum sickness-like syndrome[24, 52, 57]
2. Acute necrotizing vasculitis (polyarteritisnodosa)[24]
3. Membranousglomerulonephritis[24, 57]
4. Papularacrodermatitis of childhood (Gianotti-Crosti syndrome) [24]

TRANSMISSION

Currently, there are four recognized modes of transmission[24, 54]

1. from mother to child at birth (perinatal)

2. by contact with an infected person (horizontal)

3. by sexual contact

4. by parenteral (blood-to-blood) exposure to blood or other infected fluids.

There is considerable variation in the age at which most transmission takes place between the areas, countries and continents.

Though all body fluids which are secreted and excreted from the body contains HBsAg, only blood, vaginal and menstrual fluids and semen have been shown to be infectious[24, 52, 53, 57]. Apart from these, other body secretions show low or non-detectable levels of virus.

HBV transmission occurs by percutaneous and parenteral exposure through transfusion of blood or blood products which are not properly screened, haemodialysis, sharing of used injection needles which are not properly sterilized and injuries produced by contaminated sharp

instruments in the hospital and through permucosal exposure to infective body fluids[24, 52, 57]

Sexual and perinatal HBV transmission usually occurs from oral or vaginal mucosal exposure to blood and body fluids which are often infectious. In certain areas of south-east Asia and the far East which are considered as hyperendemic, transmission in perinatal period is common especially when HBsAg carrier mothers are also HBeAg positive.[24, 52, 57]

In groups with high HBsAg carrier rates, transmission of infection may occur between household contacts, between homosexual or heterosexual partners and in toddler-aged children.[24, 52]

The food or water contaminated by HBV will not transmit the infection. The same way, insects or other vectors has no role in transmission of HBV. But there is no convincing evidence to state that HBV infections are also airborne and faeces doesn't contain the active virus, as it is inactivated by intestinal mucosal enzymes or enzymes derived from the bacterial flora. [924, 57]

Immune globulins, albumin, fibrinolysin and heat-treated plasmaprotein fraction are considered to be safe if manufactured in an appropriate manner.

Indirect inoculation of HBV can occur due to the stability of HBV outside the human body for at least 7 days via inanimate objects when contacted with intact mucous membranes or open skin[57].HBV will retain its infectivity even in post-mortem blood.

Man is the natural reservoir for HBV[25]. But in about 35% of cases the source of HBV infection could not be identified.

There can be carriers with or without hepatitis.[57]

The failure to detect HBsAg in blood does not mean that the person is not infectious and not containing infectious virus as infectious HBV can still be present in blood of persons having no detectable levels of HBsAg[74].

The technique of reusing the same needle and syringes without proper sterilization for vaccinating many different children leads to many newer HBV infections which can be prevented otherwise.[57, 59]

People those who are likely to receive repeated transfusion due to their disease nature should be vaccinated against HBV as it is 100 times more infectious than HIV[25].

Minimal infecting dose depends upon the genotype of the infective strain. For example: A study in chimpanzees showed that one ml of

infective blood contains 102 - 109 viral copies; therefore even a microscopically blood particle can contain an infecting dose. In that study they found one positive reaction for HVB; but the epidemiological survey could not identify a positive case in the last week suggesting that the positive status of the cadaver was not known during the autopsy, nor was suspected[17]

13% of embalmers were found positive for anti HBV[42].

A survey of embalmers in the United States showed that needle stick injuries were commonly reported and that 13% of embalmers were positive for anti-HBV (about twice the rate in a blood donor comparison group)[75]

The risk of HBV infection was higher among embalmers who have worked more than 10 years with relative risk (RR) of 16.2 and those who did not routinely wear gloves with relative risk (RR) of 9.8 when compared to those who are employed in the city of Boston, RR 4.7[75].

Gershan et al, 1998 stated that exposure occur due to direct contact with non-intact skin, percutaneous injury from needles and mucous membrane exposure from blood or body fluid to eyes, nose and mouth[42].

Ball et al, 1991 stated that from needle prick, risk of infection for hepatitis B is 6-30% having no prior vaccination, hepatitis C 1.8% and HIV is 0.5%[42]

Table 3: Viral Transmission in HIV HCV and HBV

Virus	Risk of Transmission
HIV	0.2–0.5%
HCV	3–10%
HBV HBsAg-positive, HBeAg-negative HBsAg-positive, HBeAg-positive	 1–6% 22–40%

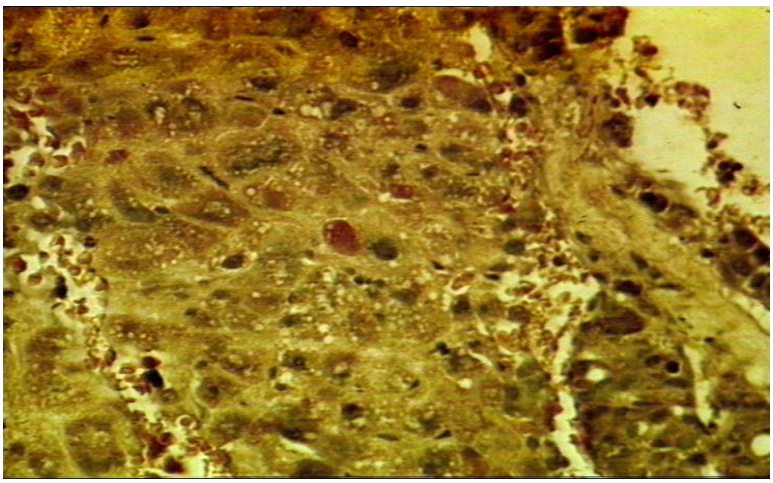
Diagnosis

The disease is usually diagnosed clinically by the symptoms which the patients manifest, biochemically by the abnormal parameters like elevated liver function test and serologically by the presence of viral markers (For eg. HbSAg).

In acute viral hepatitis, testing the patient's serum showed the anti-HBcAg (IgM), whose presence distinguishes that from a chronic infection. The panel of tests which are normally done as a screening procedure for hepatitis B virus infection includes detection of antigen and their corresponding antibodies in the blood of infected patients (HBsAg, anti- HBsAg, HBcAg, anti- HBcAg and HBeAg)

The infectious nature of the blood sample is determined by presence of HBeAg and not by HBsAg, even if present in large amounts. The HBsAg antigen which is detected in the earlier period of infection subsides with the appearance of anti-HBsAg antibodies which do not rise to a detectable level until about eight months after infection. Though the anti-HBsAg is formed as a natural immune response of the body in the early phase of infection, it forms Ag-Ab complex with HbSAg released from the cells infected with HBV and hence not detectable in the serum. This period during when neither free HBsAg nor anti-HbsAg can be detected is known as the "HBsAg window" which lasts for about six to eight months. The best tool for diagnosis of an acute HBV infection during the window is the presence of anti-HBc IgM as only a smaller quantity of HBeAg was shed from infected cells. Immunohistochemistry (IHC) also detects the presence of HBV in the given sample in the lab.

Fig 16 : Hepatitis B Positive ImmunoHistoChemistry



Screening tests for HBV infection

Hepatitis is diagnosed by testing the haematological parameters, biochemical parameters, serological and coagulation parameters in the given sample.[24, 57]

Serological parameters consists of detection of hepatitis B surface antigen, hepatitis B e antigen and/or antibody to HBsAg , antibody (IgM and IgG) to hepatitis B core antigen and antibody to HBeAg

The infectious nature of the person is determined by Serum HBsAg as it is present in both acute and chronic infections.[24, 52, 57]

Only in the presence of HBsAg, the antigens Pre-S1 and pre-S2 are seen even very early in the incubation period. The presence of HBeAg is associated with relatively high infectivity and severity of disease.[24, 57]

Anti-HBc, the first antibody to appear when demonstrated in serum indicates current or past HBV infection. Presence of IgM anti-HBc in high titre denotes acute infection. IgM anti-HBc is usually replaced by IgG anti-HBc within 6 months but it some cases of chronic hepatitis it is seen. Once the patient was infected with HBV, IgG anti-HBc is detected in his serum for his entire life.[24, 52, 57]

There is a positive correlation between the appearance of Anti-HBe after anti-HBc in serum and decreased infectivity, and if it replaces HBeAg from the circulation, it denotes that the disease is in stage of resolution.[24, 52, 57]

Anti-HBs, replaces its Ag and appears in the blood, during the resolution of disease and its presence which was observed in over 80% of patients indicate that the immunity was developed against HBV [24, 52, 57]

Those with Acute viral hepatitis who maintain constant serum HBsAg or persistence beyond 8-10 weeks after resolution of symptoms are likely to become carriers and are also at risk of developing chronic liver disease.[24]

The mutation which occurs in virus makes the antigen undetectable and diagnosis of hepatitis B difficult in a rare situation.

The Principal screening test and conclusive proof for detecting current (acute or chronic) HBV infection is the identification of HBsAg which is the first immunological marker to appear in a patient's serum and exist in high quantities in the blood.

The qualitative detection of HBsAg in human serum or plasma by an enzyme-linked immunosorbent assay test (ELISA) is a powerful and common method adopted for screening and diagnosis of HBV.

The correlation between HBV DNA replication and HBV gene expression can be obtained by examining the pathological specimens for the presence of HBV-associated antigens or particles using Immunofluorescence studies, immunohistochemistry, in situ hybridization and thin-section electron microscopy.[24]

Risk groups

The people who are at risk of contracting HBV are mentioned below[24, 57]

1. Children to children contact in day-care centre in endemic areas
2. Household with the persons infected with virus
3. The workers who engage themselves in health care and public safety
4. Patients undergoing haemodialysis and employees working in such centres[59, 76]
5. Sharing of unsterile needles for IV drug abuse, acupuncture and/or tattooing [59]
6. Sharing of unsterile equipment (medical or dental)
7. Those who live in endemic areas[77]
8. Those who travel to endemic areas[77]
9. People who engage themselves in active sex with male or female or multiple or infected persons
10. Persons with sexually-transmitted disease

11. Infants of HBsAg-positive mothers
12. Children of immigrants from high HBV prevalence areas
13. Persons requiring repeated blood transfusions

Inspite of screening procedures which are normally adopted prior to blood transfusion, the longer window period, mutant strains of HBV and low level of virus in the circulation and very high infectivity makes the recipients to acquire HBV through blood transfusion even in developed countries.

Obvious risk factors which are easily identified are not found in all patients with acute hepatitis B[58]

Treatment

At present acute hepatitis B infection has no specific treatment modality. Treatment can only be given on symptomatic basis.[24, 52]

Supportive care is the major treatment. Anti- Hepatitis B virus immunoglobulin should be given as soon as possible after the exposure of the certain patient at high risk. Immune globulin losses its effectiveness if it is given after twenty four hours of exposure to HBV.

FDA gave approval for three drugs for treating hepatitis B.

- **Interferon-alpha 2b** which is protein in nature used as natural defenses against HBV infection.
- The drug which inhibits HBV DNA polymerase (**Hepsera)** is a nucleotide analogue, which is used as a treatment modality in cases of adult survivors of hepatitis B infection which is chronic in nature.
- **Lamivudine** also given for the patients with this infection, but the main problem in this drug is development of resistance.

Even though the vaccination is highly effective in most of the patients, in practice it is very difficult to get the persons who are involved in the risk groups, because of this most of the persons who are exposed to the virus was not able to give the vaccination properly. In future, regulations should be developed for the proper immunization to the exposed individuals[56, 58]

To check the efficacy of the given vaccine, all the individuals who are vaccinated should be tested for the formation of immunoglobulins against the given antigen, after a period of 30 to 60 days after the last vaccination.

Treatment regarding the chronic hepatitis B infection should be mainly focused on (i) To prevent the circulation of virus within the body by totally the eliminating the virus (ii) To arrest the further progression of the disease occurred on the liver due to the virus (iii) Patient should be improved clinically (iv) To prevent progression to the development of carcinoma by reducing the viral markers in the circulation. Symptoms developed in the patients mainly depend on the changes in the viral markers which are developed due to the viral infection.[24, 52]

There are two classes of treatment for chronic hepatitis B:

1. Antiviral: Aimed at suppressing or destroying HBV by interfering with viral replication.[52]
2. Immune modulators: Aimed at helping the human immune system to mount a defence against the virus.

Currently, chronic hepatitis B is treated with interferons[24, 52, 57, 91].

A research is going on regarding the transfer of antibodies from the immune person to the person who is carrier of the infection, by bone marrow transplantation to totally eliminate the infection from the carrier. [24, 71]

STEPS TO MINIMIZE THE RISKS POSED BY THE INFECTED CADAVER

The degree of risk involved for the microbiology and the biomedical lab are well established, but there is no proper literature regarding the risk involved in the postmortem examination room. As there was no standard safety measures specifically designed for autopsy room based on the risks to which the persons are exposed, the safety standards which are formulated for the various clinical and investigative laboratories can be followed in mortuaries[78].

Although much has been written on how to perform necropsies on infected cadavers safely, there are remarkably few studies from which one can draw evidence upon which to base a "safe" postmortem practice. The most recent guidelines on postmortem practice published by the Royal College of Pathologists (London, UK)[79] recommend that mortuaries adopt health and safety protocols for the performance of postmortem examinations for all necropsies performed on cadavers known or suspected to be infected with a hazard group 3 pathogen.

Detailed examples of such protocols are presented in these guidelines[79]. Such detail falls beyond the scope of this review but the basic principles are presented here.

(1) **Immunization**:

The importance of immunizing the persons engaged in post mortem work is demonstrated by the comparison made between the study which was conducted among the UK health care workers in the period 1985-1988 which showed the presence of 16 cases of occupationally acquired hepatitis and the other study which showed the reduction in number of positive cases following the awareness and availability of the vaccine[80]. So it becomes mandatory to vaccinate all persons coming in contact with tissues and fluids of the dead, against tetanus[81], poliomyelitis[81], tuberculosis[82], and hepatitis B[83].

(2) **Pre-necropsy testing**:

This should be considered in cases where there is reason to suspect that the body may be infected with a previously undetected category 3 pathogen. If pre necropsy testing was positive, it becomes both the medical and social responsibility of the pathologist to make the information about the disease reach the relatives and sexual partners through the treating physician[84, 85].

(3) **Clothing**:

Contrary to the use in surgery, protective clothing worn during autopsy helps to reduce the risk of transmission of infective pathogens from the cadaver to the health care workers. The

currently recommended clothing for performing necropsies provides safety to the autopsy performers in all aspects. It includes a cap/hood, a visor, a face mask with micro filter, surgical shirt and trousers, waterproof boots with steel toecaps, a full length gown, a waterproof apron and at least one pair of gloves [40, 83, 84, 86, 87, 88, 89,]. Some pathologists may form a waterproof seal[90]. It may be hazardous also by a reduction in field of vision and communication[91]. O'Briain[92] observed the increased frequency of cuts when compared to needle stick injuries in autopsies. There is inverse correlation between the frequencies at which the injury occur while during autopsy and the experience of person performing the procedure.

Mast et al demonstrated the 63 % reduction in transfer of blood through needle stick injury by sutures and 86% reduction in transfer of blood through needle stick injury by hollow needles which were significantly achieved by wearing surgical gloves[93].

Weston and Locker[40] stated that 31.8 % of the glove punctures which occur during autopsy procedures were not noticed at the time of puncture by the technician wearing the gloves. Though some authors felt it was an unnecessary expense to wear double gloves, it should be noted that the perforation rate of inner glove is reduced by the habit of wearing double gloves. According

to few authors, the gloves should be changed frequently during the post mortem examination irrespective of whether it was damaged or not. As it was observed that multiple perforations seen in the base of ring finger where normally we wear ring, it was advised that the glove should be donned only after removing the ring in order to reduce the frequency of glove perforations[94]. The gauntlets made of metal mesh[90, 95, 96] or **Kevlar**[97] can be used on non-dominant hand to provide additional protection against cuts but not against needle stick injuries. Though the usage of gauntlets had an added advantage it gives a cumbersome feel and the autopsy surgeon cant able to feel the texture and consistency of the organs as it acts as an interface between the hand and the organ and restrict the usage of hand to a maximum[90, 95].

Weston and Lober[40]et al. have documented that among the 8% glove punctures that occur during the course of post mortem examination, about 33 % was not recognized at the time of puncture which makes the pre existing injuries in the hands of autopsy performers to come in contact with infectious pool of blood and body fluids for a longer period. Although gloves are impermeable to HIV and HBV infection, punctured gloves are not. The most important way of reducing the infection associated with

glove puncture is frequent change of gloves and hand washing throughout the procedure and at the end of procedure[40]

(4) **Reduce aerosol formation**:

This is essential for reducing the risk of acquiring airborne infections such as tuberculosis[98] and enteric pathogens[99]and for necropsies on patients suspected of having HIV or TSE. It should be realized that most airborne bacteria in mortuaries are derived from the skin of the staff present in the mortuary[99, 100]. The aerosol transmission and odour can be reduced by the use of down draught ventilation tables[18, 99, 100]. The risk of aerosols is high with use of power saws[99.] The intestine should be opened under water to reduce the risk of aerosol[81, 90, 95, 96.] As splashing of water and body fluids increase the formation of aerosol, avoid using water sprays at high pressure[81, 99]. The clean plastic bags can be placed over head at the time of removing the brain to reduce spread of aerosols[101]. The advantage in eviscerating the infected body, organ by organ when compared to Letulle technique of en bloc removal was observed by some authors[91]. Instead of plastic bags, specific tents have been designed to reduce the aerosol by covering the head and neck of cadaver[101].

(5) **Equipment**:

Those workers who are at frequent exposure to sharp objects (Needles or Instruments) and blood are at increased risk of acquiring hepatitis B virus infection[102]. Only minimum equipment that was kept in a clear view should be used. Avoid instruments with pointed ends[88,91]. Instruments (especially sharps) should be transferred from one's hand to other through a vehicle, commonly used is kidney tray[90]. Use disposable instruments, whenever possible, in high risk cases. Non-disposable instruments needs to be disinfected for longer periods[90].

(6) **Circulators**:

All high risk autopsy cases need not performed in a specialist mortuary, but make sure only minimum persons are present during the procedure[91, 103]. Therefore, in practice, the pathologist, anatomical pathology technician and circulator will be in the autopsy rooms, [90, 104] first two persons will be in contact with the cadaver and collect the samples, third one will help in other clerical work associated with such specimens. The circulator constantly monitors the other two persons involved in autopsy and ensures that the safety is maintained inside the mortuary as per the health and safety guidelines[104].

(7) **Safe sharps practice**:

As the sharp ends of the instruments used by the autopsy performers and the sharp ends of the fractured fragments of bone and unanticipated sharp objects that may present within cadaver may come in contact with the personnel any time during the procedure, safety should be look into by all means. At any cost avoid dissecting the cadaver blindly. The safety acquired through the practice of evisceration in post mortem examination was evaluated by only one study.

Walker etal found that increased risk of injuries occurring by sharp edge of ribs which was produced by using rib shears is better avoided by using an electric saw, but it has an disadvantage of increasing the risk of aerosol and vibration induced white fingers (when used for long time) in the persons handling them[105, 106]. In considering the safety of the persons handling the dead bodies, some authors recommend the use of staples or tissue adhesives to close the post mortem incision wound and reconstruct the body instead of suturing, as the gauntlets we use also won't protect us from suture needles. Some authors even say to leave the body as such in a leak proof body bag and seal it[84, 91]. Regarding the best way of handling the infected body, till now no proper studies or no standard guidelines were available.

Herbst et al, 2009 stated that the Universal precautions for blood and body fluids and enteric precautions should be followed[42].

Other personal protective equipment like eye wear, gowns and masks are only required when large splashes of blood are anticipated.

Curti and Biran, 2001 stated that the hands should be washed after handling the cadaver and before eating and all equipment washed with disinfectant[42].

Rischetelli et al, 2001 and **De ville,1980** in his study stated that the body bags which are used, can also reduce the infection and are useful for transportation of cadavers which are badly damaged. But the body bag reduces the rate of cooling and increase rate of decomposition especially in hot climate[42].

PROPHYLAXIS

Hepatitis B vaccination prevents infection and is 70-80% effective within a week of exposure. Therefore while dealing with cadavers, covering of cuts or lesions with water proof dressings, careful cleansing of any injuries sustained during embalming, particularly use of appropriate protective clothing will greatly reduce the risk of acquired infection[34.]

IMMUNISATION SCHEDULE

- 1st dose at elected date
- 2nd dose should be given 4 to 10 weeks after the 1st dose
- 3rd dose should be given 1 to 5 months after the 2nd dose

Booster dose should be given particularly for all people at risk, when the anti-HBs antibody titre falls below 10 IU/L. After the 0, 1, 2 months of primary immunization schedule a booster dose is recommended 12 months after the first dose. After 8 years the next booster may be required. After the 0, 1, 6 months primary immunization schedule a booster dose may be required after 5 years after the primary course.

METHODOLOGY

(MATERIALS & METHODS)

METHODOLOGY (MATERIALS & METHODS)

The samples were tested blindly that the identity of the individual was unknown. The samples were collected via cardiac chamber at the time of autopsy.

Reagents And Materials Provided with Human HBsAg ELISA Kit:

Microwell Plate:

The blank microwell strips fixed on white strip holder is sealed in aluminum pouch with desiccant. Each well contains monoclonal antibodies reactive to HBsAg (anti-HBs). It can be broken and use separately if the sample volume is small. The unused wells or strips is placed back in the bag provided and maintained a 2-8°C.

Negative Control:

Protein-stabilized buffer yellowish in color and tested non-reactive for HBsAg. It is identified by green screw cap.

Positive Control:

HBsAg diluted in protein-stabilized buffer which is red in color. It is identified by red screw cap

HRP-Conjugate:

Horseradish peroxidase - conjugated anti-HBs antibodies which is also a red-colored liquid. To distinguish from positive conjugate which is also of same color, it is kept in a white vial with red screw cap

Specimen Diluents:

It is Serum base, casein and sucrose solution in green color placed in a vial with blue screw cap.

Wash Buffer:

Colorless liquid filled in a clear bottle with white screw cap. PH 7.4, 20x PBS. The concentrate must be diluted 1 to 20 with distilled/ deionized water before use. Once diluted, stable for one week at room temperature, or for two weeks when stored at 2-8°C.

Chromogen Solution A:

Urea peroxide solution which is a colorless liquid. It is kept in a white vial with green screw cap.

Chromogen Solution B:

TMB solution (Tetramethyl benzidine dissolved in citric acid) a colorless liquid. It is kept in a black vial with black screw cap.

Stop Solution:

Diluted sulfuric acid solution (0.5M H2SO4) a colorless liquid in a white vial with white screw cap.

Note:

Microwell Plate, Negative Control, Positive Control and HRP-Conjugate, Chromogen Solution A, Chromogen Solution B, Stop solution will be stable only for one month after it was opened only when the optimal temperature is maintained. All are ready to use as supplied

Principle of the Test:

Human HBsAg ELISA Kit uses antibody "sandwich" ELISA method for detection of HBsAg, in which, polystyrene micro well strips are pre-coated with monoclonal antibodies specific to HBsAg.

a) Add Patient's serum or plasma sample to the microwells
b) Polystyrene micro well strips are pre-coated with monoclonal antibodies specific to HBsAg
c) During incubation, if HBsAg is present in the sample, the specific Ag Ab complex formed is captured on the solid phase.

d) Then the second antibody conjugated the enzyme horseradish peroxidase (the HRP-Conjugate) directed against a different epitope of HBsAg is added into the wells.

e) During the second incubation step, these HRP-conjugated antibodies will be bound to any anti-HBs-HBsAg complexes previously formed during the first incubation, and the unbound HRP-conjugate is then removed by washing.

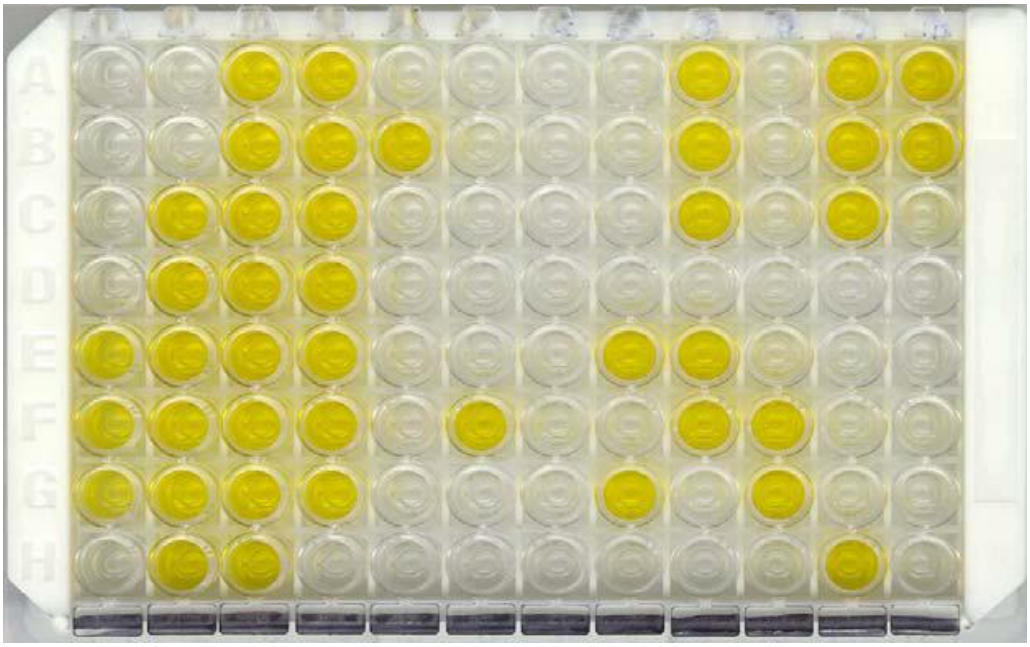
f) Chromogen solutions A and B are added to the wells.

g) In presence of the antibody-antigen-antibody (HRP) "sandwich" immunocomplex, the colorless chromogens are hydrolyzed by the bound HRP-conjugate to a blue-colored product.

h) The blue color turns yellow after stopping the reaction with sulfuric acid.

The amount of color intensity can be measured by determining absorbance using 450nm as reading wavelength with 620-690 nm reference wavelength which is proportional to the amount of antigen captured in the wells, and to its amount in the sample respectively. Wells containing samples negative for HBsAg remain colorless.

Fig 17: Diagram of Generic 'Antigen Sandwich' Elisa for Completed Generic Elisa Assay



A – Blank

B C D - Negative controls

E F - Positive controls

G - Sample 1 Positive

H - Sample 2 Negative

Table 4 : Interpretation of the HBV serological markers

Tests	Results	Interpretation
HBsAg anti-HBcAg anti-HbsAg	Negative Negative Negative	1. No HBVexposure 2. Not vaccinated

HBsAg anti-HBcAg anti-HbsAg	Negative Positive Positive	1. Infected with HBV 2. And Immune
HBsAg anti-HBcAg anti-HbsAg	Negative Negative Positive	1. Vaccinated and Immune 2. Not infected
HBsAg anti-HBcAg anti-HBcAg IgM anti-HbsAg	Positive Positive Positive Negative	Acute HBV infection
HBsAg anti-HBcAg anti-HBcAg IgM anti-HbsAg	Positive Positive Negative Negative	Chronic infection
HBsAg anti-HBcAg anti-HBsAg	Negative Positive Negative	1. Recovery from Acute HBV infection 2. HBV carrier 3. Window period 4. False positive to anti-HBcAg

Subject Selection:

Study would be conducted on those cases, coming for Medico Legal autopsy to the Institute of Forensic Medicine, Madras Medical College, Chennai - 600003. The identity of the individual should not be disclosed in any part of the study.

Inclusion Criteria:

All dead bodies subjected for autopsy

Exclusion Criteria:

All decomposed dead bodies subjected for autopsy

LIMITATIONS OF THE TEST:

1. Only un-pooled human serum or plasma can be used
2. HBV infection cannot be excluded without considering the other evidences for the same by taking only the negative HBsAg obtained

PERFORMANCE CHARACTERISTICS

(i) Diagnostic specificity: 99.58%

(ii) Diagnostic sensitivity: 100%

ANALYSIS AND RESULTS

ANALYSIS AND RESULTS

Out of the 515 samples tested, Males occupy predominant number of cases, accounts for about 86.6% of study samples, whereas female constitute only 13.2% of the study sample.

Of the 515 samples tested, HbsAg were detected in 18 samples (3.5%) using ELISA kit. All the positive cases were not previously known to have HBV-infection. Data such as demographics, cause of death, brought dead or treated, post mortem interval and positivity for HbsAg are recorded.

Out of the 18 positive samples, 13 were male and 4 were female and 1 transgender.

Table 5: Sex distribution among the study sample

Sex	No. of Cases	Percentage of Cases	No. of positive cases	Percentage of positive cases
Male	446	86.6%	13	2.9%
Female	68	13.2%	4	5.9%
Transgender	1	0.2%	1	100%
Total	515		18	3.5%

Among 515 cases analyzed, predominant numbers of cases are male patients (86.6%). In this study, out of the 18 positive cases, thirteen cases were positive among the males (72.2%) with 2.9 % of overall positive cases and four cases were positive among the females (22.22 %) with 5.9 % of overall positive cases, one case was found in transgender (5.5 %) with 0.2 % of overall positive cases. In this study the prevalence of HBV is 100 % in Transgender as only one case was tested and it came as positive which is followed by male population.

Fig 18: Sex distributions among the study sample

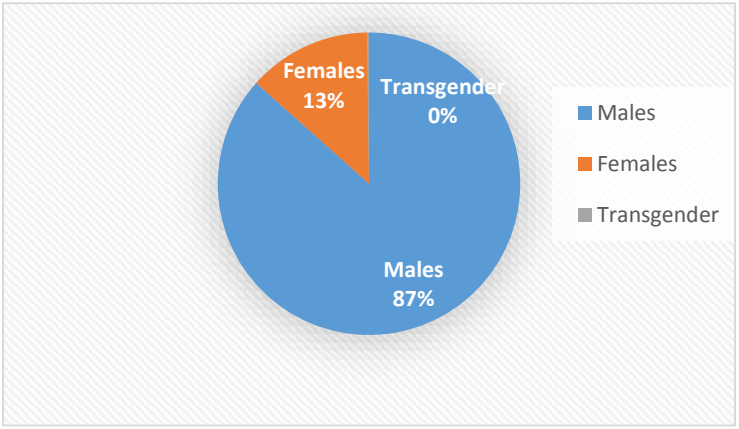


Fig 19 : Positivity among both the sexes

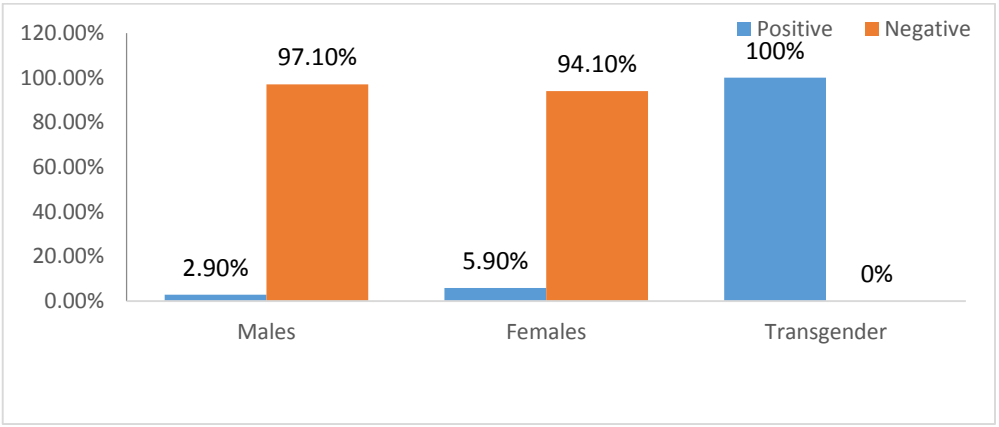


Table 6: Age distribution among the study sample

S.no	Age distribution	No. of sample	Percentage of samples	No. of positive cases	Percentage of positive cases
1.	0 - 10 years	7	1.36%	0	0%
2.	11 - 20 years	30	5.82%	0	0%
3.	21 - 30 years	121	23.5%	6	4.95%
4.	31 - 40 years	87	16.9%	4	4.59%
5.	41- 50 years	121	23.5%	5	4.13%
6.	51- 60 years	87	16.9%	3	3.45%
7.	61- 70 years	45	8.74%	0	0%
8.	71- 80 years	15	2.91%	0	0%
9.	81- 90 years	2	0.4%	0	0%
10.	Total	515		18	

Among 515 cases analyzed, predominant number of cases falls under age group between 21 to 30 and 41 to 50, followed by age group between 31to 40 and 51 to 60. In this study, Out of the 18 cases, six cases were positive in the age group 21-30, five cases were positive in the age

group 41-50, four cases were positive in the age group 31-40 and three cases were positive in the age group 51-30 indicating that highest seroprevalence was in the age group 21-30.

Fig 20: Age distribution among the study sample

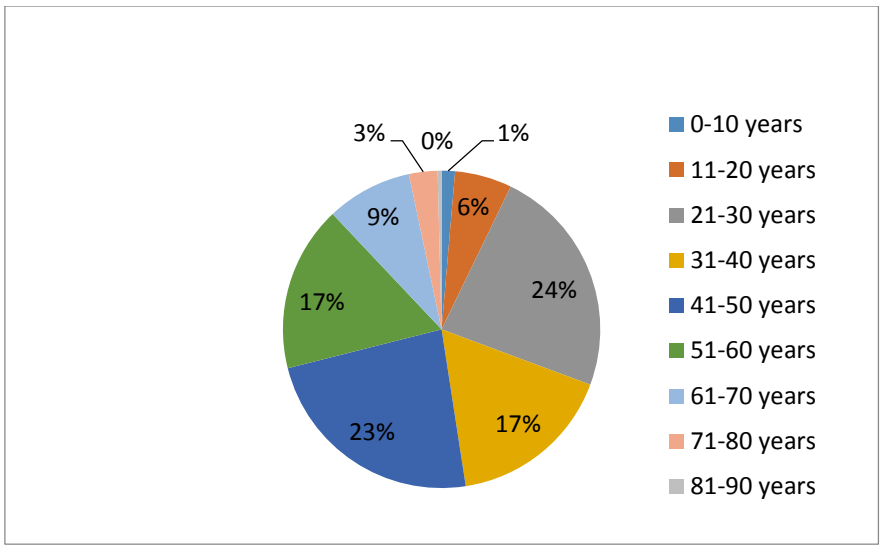


Fig 21: Positivity percentage among the age distribution in study sample

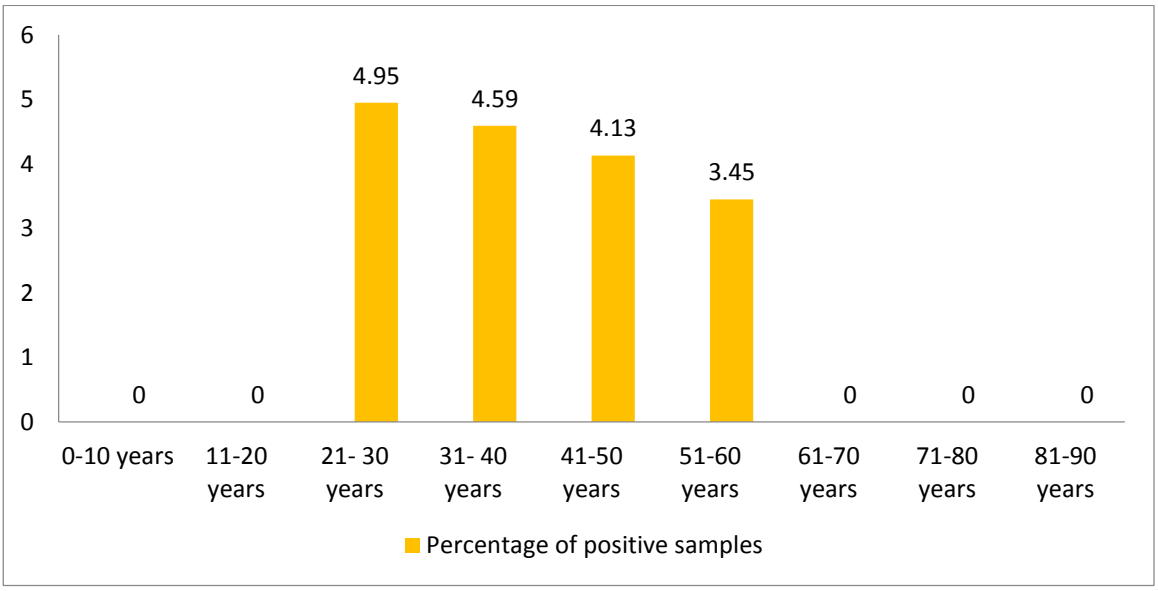


Table 7 : Distribution of manner of death among the sample collected and positive cases of the study sample

S.No	Cause of death	No. of cases	Percentage of cases	No. of positive cases	Percentage of positive cases
1.	Natural cause	53	10.3%	4	7.56%
2.	Road traffic accident	265	51.46%	3	1.13%
3.	Train traffic accident	34	6.6%	2	5.88%
4.	Hanging	19	3.69%	3	15.79%
5.	Fall	59	11.46%	1	1.7%
6.	Murder	17	3.3%	3	5.13%
7.	Poisoning	39	7.57%	2	5.13%
8.	Other causes*	29	5.63%	0	0%
9.	Total	515		18	

* (Snake Bite – 10, Electrocution – 8, Suspicious Death - 3, Scorpion Bite - 1**,** Industrial accident - 1, Drowning - 3, Accident - 2, Burns - 1)

Among 515 cases analyzed, predominant number of cases falls under the category of Road Traffic Accidents (51.46%), followed by Fall from height (11.46%), Natural cause (10.3%), Poisoning (7057%) and others. In this study, Out of the 18 positive cases, four cases were positive in the Natural cause, Three cases were positive in the RTA, Hanging and

Murder cases, two cases were positive in TTA and Poisoning and one case was positive in the Fall from height. Even though Road traffic accidents constitute the majority of samples collected (51.46%) and Natural cause (with 7.56 % of seroprevalence) constitutes 22.23 % of overall positive samples, highest seroprevalence was noted in case of Hanging with 15.79% (Three cases were positive out of nineteen cases) with 16.67% of overall positive samples (Three out of eighteen cases).

This shows that HBV screening is of great importance among the cases we receive for autopsy irrespective of manner of death. Hanging and natural cause in our study showed a relatively higher prevalence of HBV infections compared with other manners of deaths.

Fig 22: Distribution of manner of death among the study sample

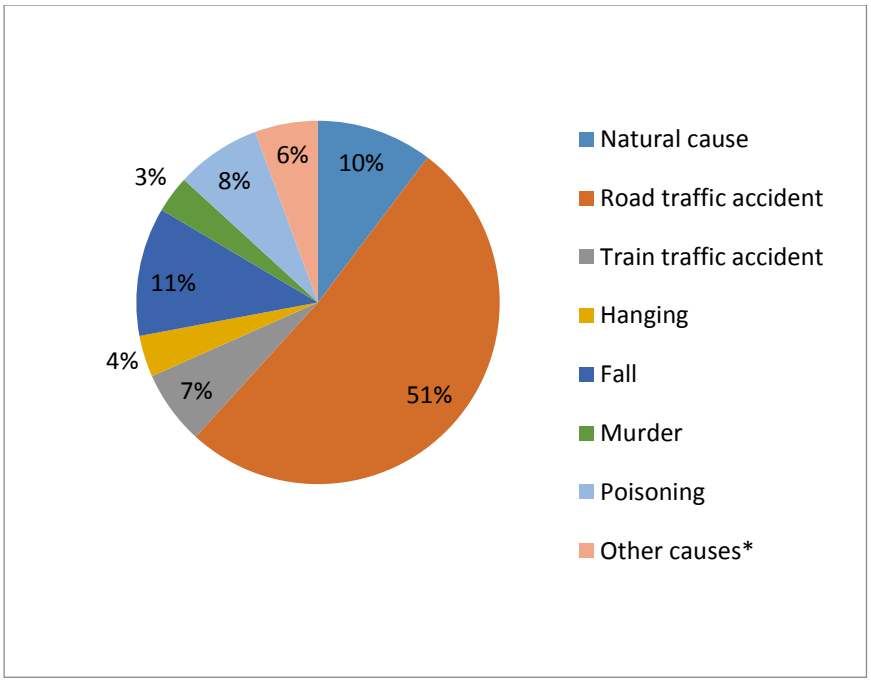


Fig 23: Distribution of percentage of positive cases of the study sample among the manner of death

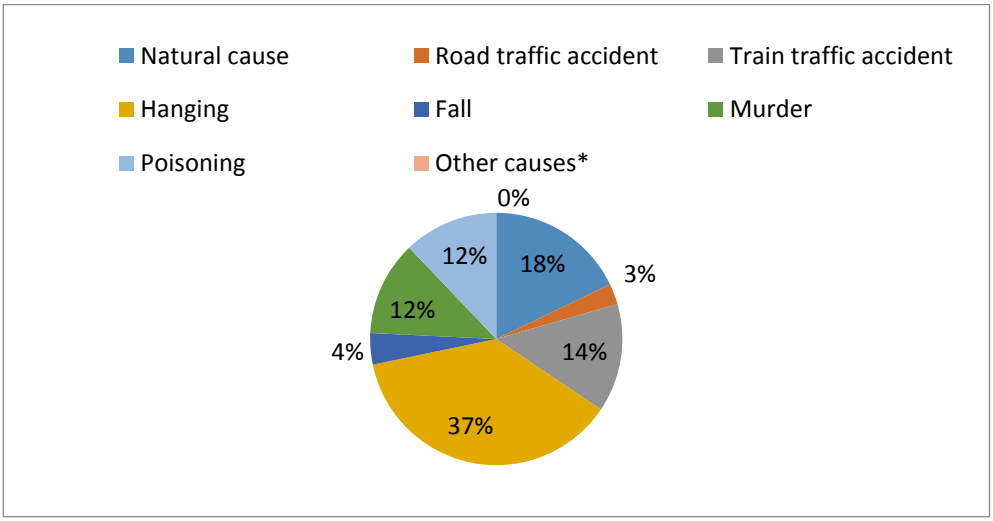


Table 8: Distribution of Brought dead cases among the study sample

S.No	Cases	No of Cases	Percentage of cases	No of Positive cases	Percentage of positive cases
1.	Brought Dead	91	17.67%	12	13.2%
2.	Hospital Treated	424	82.33%	6	1.42%
	Total	515		18	

Among 515 cases analyzed, predominant number of cases falls under the category of Hospital treated patients (82.33%). In this study, Out of the 18 positive cases, twelve cases were positive among the brought dead category (13.2%) with 66.67 % of overall positive cases and six cases were positive among the hospital treated category with 33.33% of overall positive cases.

This shows that HBV screening is of great importance among the cases we receive for autopsy as their HBV status was not known (66.67% were brought dead with no details regarding history of exposure or blood transfusion or HBV/HIV status)

Fig 24: Distribution of brought dead and treated case among study sample

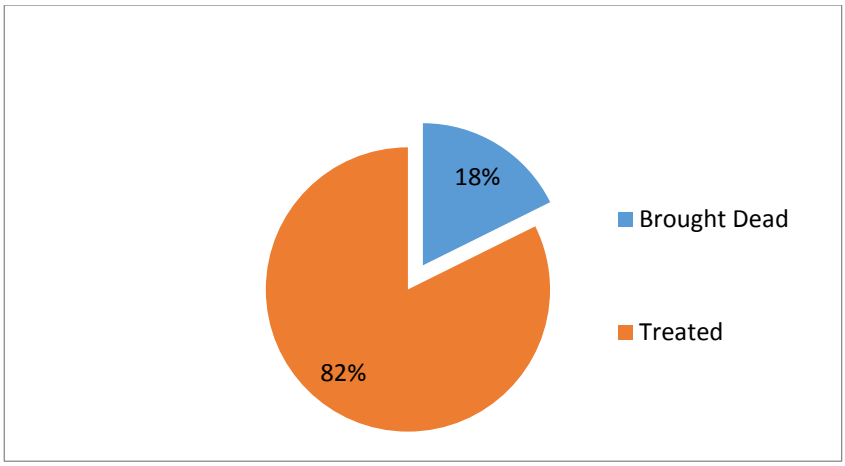


Fig 25: Distribution of Positive case among the brought dead and treated case in the study sample

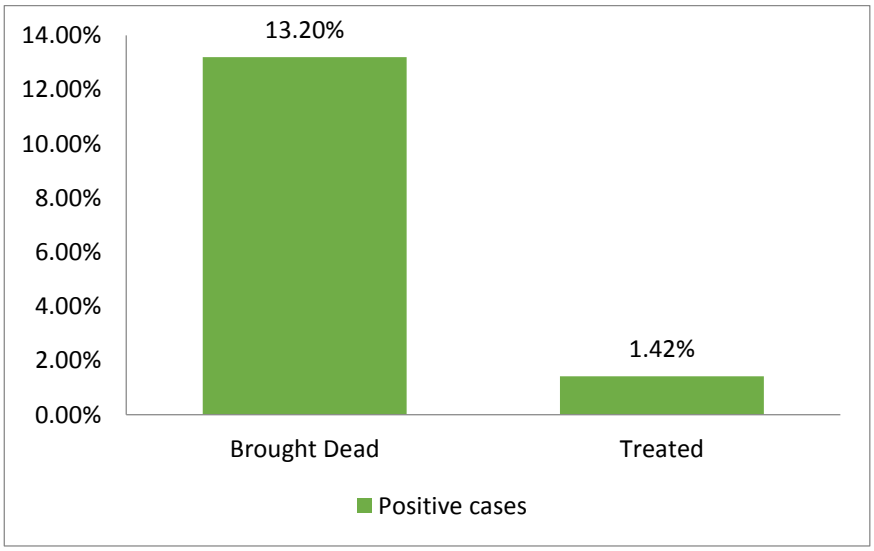


Table 9: Distribution of Post mortem interval among the study sample

S.No	Post mortem interval	No. of Samples	Percentage of samples	No. of Positive cases	Percentage of positive cases
1.	< 6 Hours	26	5.04%	1	3.85%
2.	6 – 12 Hours	101	19.6%	2	2%
3.	12– 24 Hours	263	51.1%	13	4.94%
4.	24– 36 Hours	101	19.6%	2	2%
5.	>36 Hours	24	4.7%	0	0%
	Total	515		18	

Among 515 samples analyzed, predominant number of samples was collected between 12 to 24 hours (51.1%), followed 6 to 12 hours (19.6%) and 24 to 36 hours (19.6%). In this study, out of the 18 positive cases, thirteen cases were positive among the samples collected 12 to 24 hours after death (positive percentage of 4.94%) with 72.22 % of overall positive cases.

The samples collected upto 36 hrs after death showed positive results for HbsAg. This shows that HBV screening is of great importance among the cases we receive for autopsy as HbsAg was detected even in

the post mortem blood samples and hence HBV can be transmitted from cadaver to health care workers

Fig 26: Distribution of Post mortem interval among the study sample

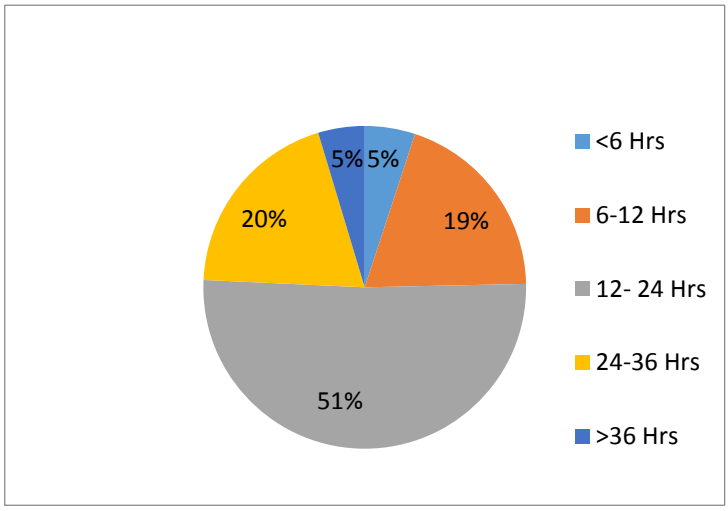


Fig 27: Distribution of Positive percentage among Post mortem interval in the study sample

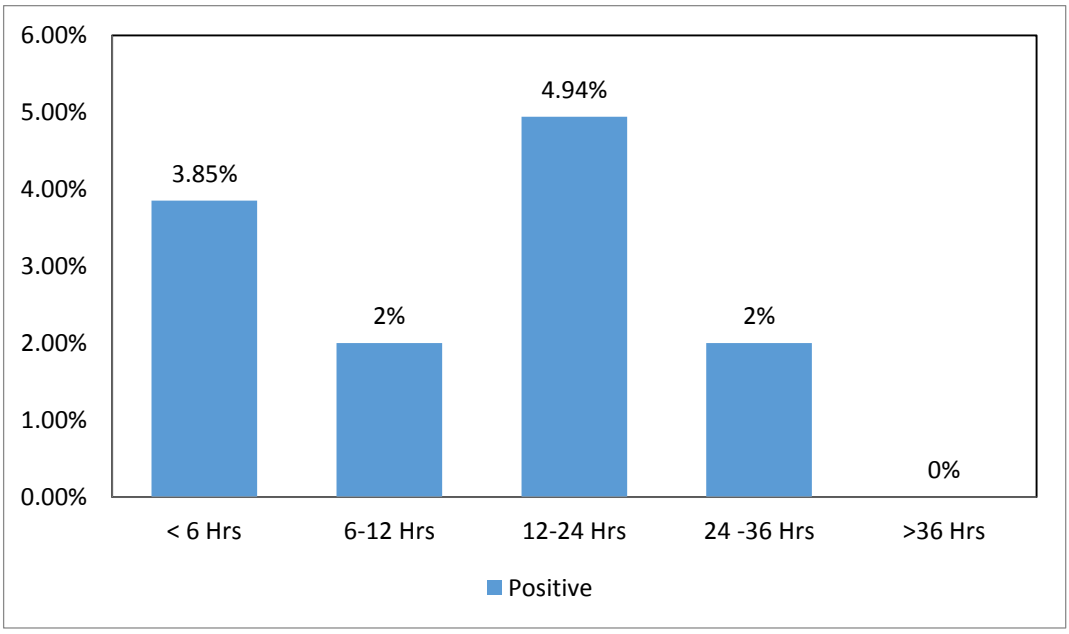


Table 10: Distribution of nature of occupation among the study sample

S.No	Occupation	No. of Cases	Percentage of cases	No. of Positive cases	Percentage of positive cases
1.	Coolie	248	48.12%	6	2.42%
2.	Private	72	14%	1	1.4%
3.	Driver	38	7.4%	4	10.53%
4.	House wife	27	5.24%	1	3.7%
5.	Security	7	1.4%	1	14.3%
6.	Merchant	5	1%	1	20%
7.	Cleaner	2	0.4%	1	50%
8.	Fish man	3	0.6%	1	33.3%
9.	Sex worker	1	0.2%	1	100%
10.	NK	5	1%	1	20%
11.	Other occupation*	107	20.78%	0	
	Total	515		18	

* **(**Student – 24, Farmer – 19, Painter -12, Govt – 11, Mason – 7, Tailor – 7, Business – 4, Carpenter- 4, Retd – 4, Kid – 3, Electrician – 2, Mechanic – 2, Cashier – 1, Cine field – 1, Cook – 1, Gold smith – 1, Butcher – 1, Lawyer – 1, Broker – 1,Plumber – 1,)

Among 515 cases analyzed, predominant number of cases falls under the category of Coolie (48.12%) followed by Private Employee (14%) and Driver (7.4%). In this study, Out of the 18 positive cases, Six

cases were positive in Coolie, Four cases were positive in Drivers and other eight positive in Private employee, House wife, Merchant, Security, Cleaner, Sex Worker, fish man and Not Known category. Even though Coolie constitute the majority of samples collected (48.12%) with 2.42 % positive percentage and constitutes 33.3 % of overall positive samples, highest seroprevalence was noted in case of Sex worker with 100% (One case tested which was found to be positive) with 5.56% of overall positive samples (One out of eighteen cases).

This shows that HBV screening is of great importance among the Coolie and commercial sex workers which showed a relatively higher prevalence of HBV infections compared with other occupations

Fig 28: Distribution of Occupation among the study sample

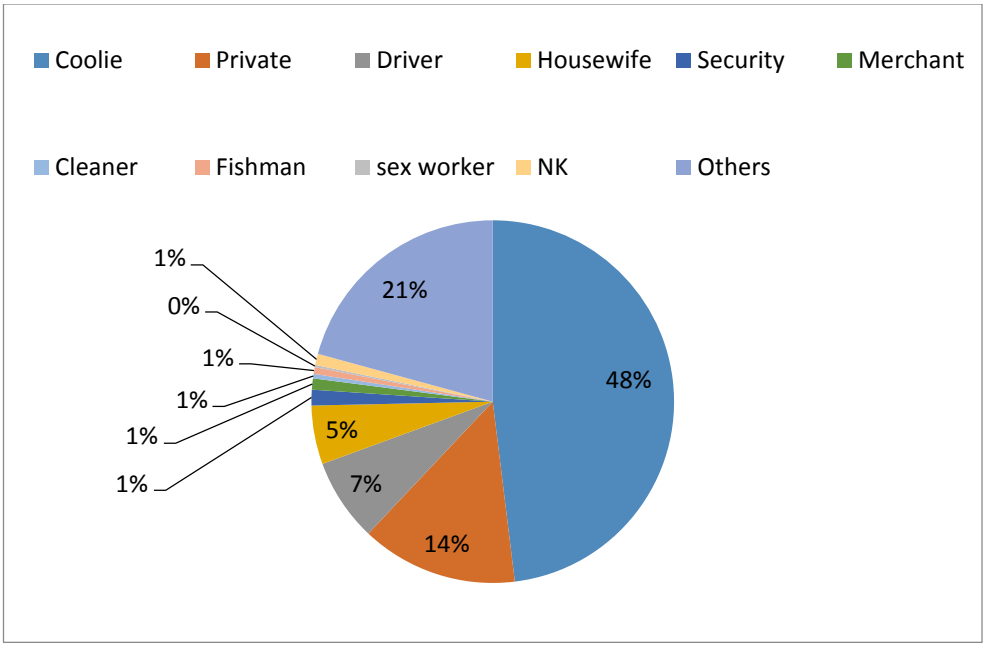


Fig 29: Distribution of Positive percentage among the various occupational groups in the study sample

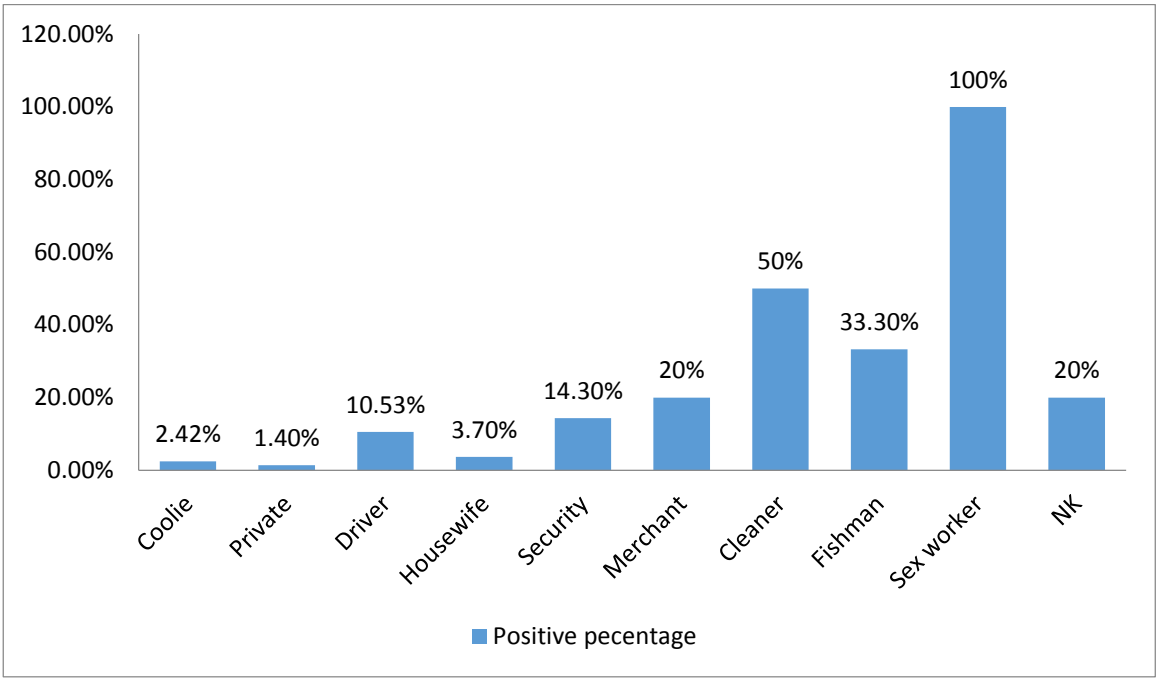


Table 11: Distribution of marital status among the study sample

S.No	Marital Status	No. of Cases	Percentage of cases	No. of Positive Cases	Percentage of positive cases
1.	Married	402	78.1%	13	3.23%
2.	Single	108	21%	4	3.73%
3.	Not Known	5	1%	1	20%
	Total	515		18	

Among 515 cases analyzed, predominant number of cases falls under the Married category (78.1%) followed Single (21%). In this study, Out of the 18 positive cases, thirteen cases were positive in married

population, four cases were positive in Singles. Even though Married population constitute the majority of samples collected (78.1%) with 72.33 % of overall positive samples, Both Married and Single constitutes seroprevalence of about 3% positive percentage with unknown category having highest seroprevalence of 20% (One in five cases positive) with 5.56% of overall positive samples (One out of eighteen cases).

This shows that HBV screening is of great importance in the Spouse and children when the deceased was found to be positive in autopsy samples. Dead bodies brought as unknown without no relatives showed a relatively higher prevalence of HBV infections compared with other category. Hence unless clearly indicated, unknown bodies should not be subjected for autopsy as a routine procedure.

Fig 30: Distribution of Marital status among the study sample

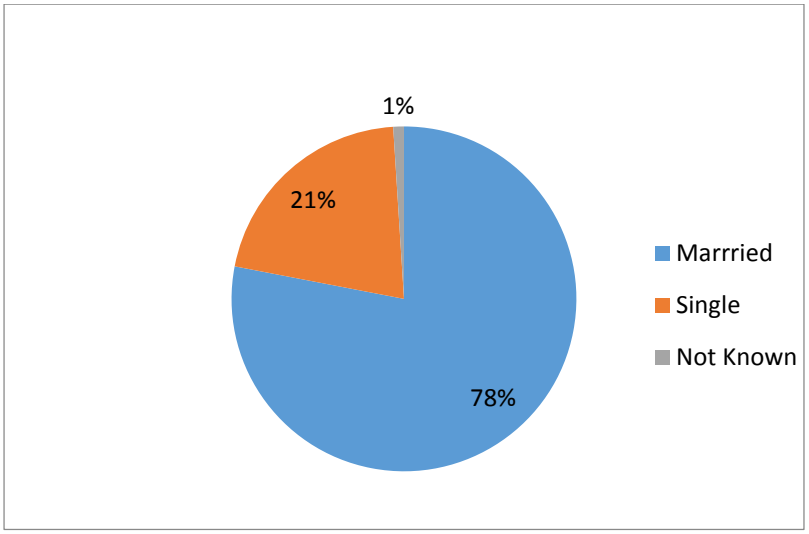
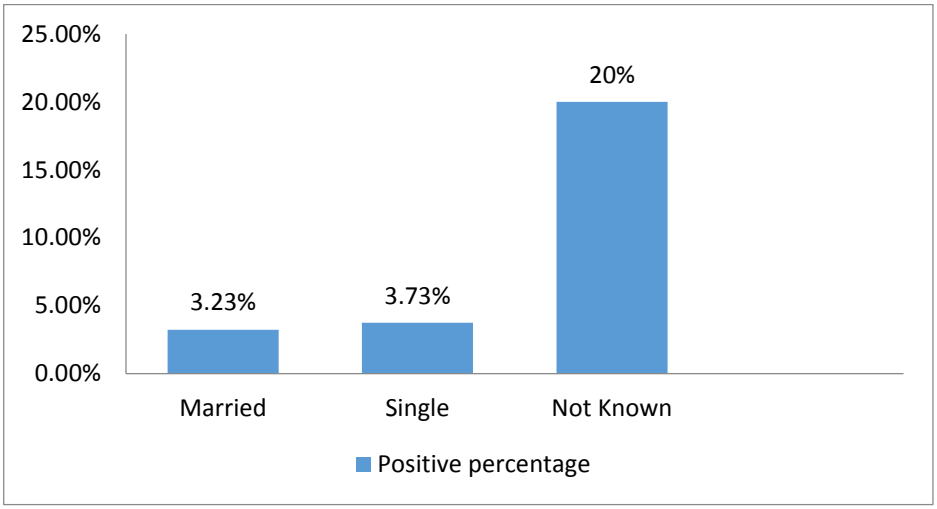


Fig 31 : Distribution of positive percentage among the marital status in the study sample



DISCUSSION

DISCUSSION

Hepatitis B virus had an increased morbidity and mortality among the mortuary workers due to its high frequency among the deceased and longevity of the virus[14.] Amongst all the parenteral viruses, Hepatitis B virus has the highest transmissibility rate with a rate of about 100 times greater than HIV

Carolin et al suggested that post-mortem blood samples should be collected within 24 hours. **Challine et al. (2006)** recommended 12 h maximum time for drawing post-mortem blood samples. In my study HbsAg was detected in the post mortem blood samples upto 36 hours.

In 2012 in India about 119,000 cases of all cause of viral hepatitis were reported. The estimated burden of chronic HBV infection in the South-East Asia region was 100 million. After HEV, HBV is the second most common cause of acute viral hepatitis in India with over 40 million HBV carriers. India is considered to have an intermediate level of HBV with 3.7% point prevalence.

In India, every year one million populations are at risk and about 100,000 die from HBV infection. Though HDV infection is not very common in India it is observed in 10% to 20% of HBV positive patients.

Genotypes A and D of HBV, and genotypes 1 and 3 of HCV are more prevalent in India.

In India epidemics due to the practice of unsafe injection among injecting drug users and healthcare workers caring the infected people 46% of hepatitis B carriage and 38% of HCV infection were documented.

In India, HBV infection was transmitted perinatally in about 10% of cases when the mother is only hepatitis B surface antigen (HBsAg) positive but when the mother is positive for both HBsAg and hepatitis B e-antigen (HBeAg) the transmission rate increases to 90%

Universal immunization against hepatitis B was introduced in India in 10 states in the year 2002 and in 2011 countrywide. A pentavalent vaccine which was introduced recently in some states gives protection against HBV also.

A simple, reliable, and rapid test to detect HBV infection can be useful in the mortuary because it is not possible always to get the complete and correct information about all of the risk factors before commencing the postmortem, because of the social and cultural restrictions. Therefore the lack of a known history of such a risk factor does not equate with the nonexistence of such risk factor. Hence testing for HBV in medico-legal autopsies will identify carriers in whom BIV status was not previously known.

Li et al[30] reported 23% prevalence of hepatitis B in forensic autopsy performers

Plessis et al[41] reported 8% prevalence of hepatitis B in forensic autopsy performers

In this study, the eighteen positive cases out of 515 cases were not known to have HBV which means they are clinically undetected for HBsAg. This indicates that that people at high risk are clustered in the medico-legal autopsy series.

There are many studies that have estimated the survival of the HBV postmortem. HBV survive outside the human body for upto 7 – 10 days and can withstand drying for atleast a week. When stored at 30-32 degree Celsius, HBV retains its infectivity for atleast 6 months and when frozen at -15 degree Celsius, its infectivity will be retained for upto 15 years. During the early phase of infection, the viral load in blood and other body fluids may be very high. This suggests the need for efficient protection of autopsy personnel's during all autopsies.

The testing of postmortem sera for HbsAg will be a most reliable measure of antemortem HBV infection. HbsAg and anti -HbsAg have been shown to remain detectable in postmortem serum stored for relatively long periods of time. Furthermore, it has been demonstrated that by assessing the post mortem changes in the serological parameters Kitchen and Newham established the post mortem stability of serological markers upto 24 hours after death. East lund and Schuller demonstrated

that the post mortem stability of the stability of the antibodies was similar when compared to the ante mortem screening done in live donors.

In general, proteins such as the globulins that comprise antibodies may not be affected by postmortem decomposition, hemolysis, or bacterial contamination[23, 30]. In this study, it was estimated that the postmortem interval was ranging from 6 hours to 36 hours for detection of HbSAb.

Postmortem viral level is influenced by several factors like viral burden of death, viral strain, pre-mortem antiviral therapy and temperature maintained in the mortuary. HBV is transmitted from person to person when they come in contact through blood, vaginal fluid, semen, breast milk, CSF, amniotic, pericardial and synovial fluids. Others such as saliva, tears, urine unless they found to be contaminated with blood in adequate volume are not implicated in the transmission of HBV.

British clinical laboratories conducted a study in 1970 - 1989 and established that the rate of infections acquired through handling of laboratory equipments and specimens was highest in mortuary workers. Weston and Lober have recognized that about 8% surgical gloves get punctured during post-mortem and nearly one third of that was undetected by the pathologist, thus causing the infectious blood to be bathed in any preexisting hand injuries for a prolonged period of time[40].

In the present study, the samples were tested blindly, that the identity of the individual was unknown. This study includes all types of forensic autopsy cases representing the general population. Samples of blood were collected from 515 autopsy cases at Rajiv Gandhi Government General Hospital. All the samples were tested for HbSAg using standard Human HBsAg ELISA kits which has a sensitivity of 99.58% and specificity of 100%.

Out of 515 cases, there are eighteen positive cases and thirteen positive cases were male, four positive cases were female and one case was transgender. The presence of 18 positive cases among 515 cases, though statistically insignificant is higher than many other voluntary screening programs (The seroprevalence of HIV, HBV, HCV and syphilis was found to be 0.154%, 0.887%, 0.101%and 0.22% respectively in voluntary blood donors **(A study on Sero prevalence of HBV, HCV, HIV and syphilis among blood donors at a tertiary Care Teaching Hospital in Western India)**. In many voluntary screening programs there are people who abstain from testing, which does not affect in this study. Thus, screening in medico legal autopsy is a sensitive indicator epidemiologically.

Many studies conducted for detecting the HbsAg in the autopsy population were classified the deceased as “known risk” and “no known

risk" groups based on the history such as infection, drug addiction, and sexual contact but that was not reliable and leads to misclassification. So, in this study such data about the risk factors were not collected.

There are two opinions concerning HBV in autopsies. One school of thought maintains that all autopsies should be carried with universal precaution which is almost impracticable in a developing country. The second thought recommends pre autopsy testing for HBV by rapid test kits in the mortuary by taking blood samples from the dead body. If the result is positive, then universal precautions to be observed while conducting autopsies.

LIMITATION OF THE STUDY: The obstacles for postmortem testing of blood and body fluids include hemolysis, autolysis, bacterial contamination, and loss from decomposition. These obstacles are enhanced by prolonged postmortem intervals. However, immunoglobulins, are considered less likely to be affected by decomposition, hemolysis and bacterial contamination.

This screening test for the HbsAg may not be sensitive if the person was in the window period

CONCLUSION

CONCLUSION

The health care professionals who are involved in the postmortem work are exposed to a greater risk of occupational health hazard due to higher prevalence of various infectious diseases among the general population. It is wise to practice the Universal Precautions in almost all dead bodies considering it as infectious as it becomes practically impossible to know the medical status of each and every body subjected for autopsy.

The autopsy based occupational health hazards can be reduced by focusing on improvement in assessment, training, education, personal protection, autopsy techniques and autopsy room.

Health care professionals involved in postmortem practice should be aware of the hazards and risks associated with the cadaver, instruments they handle and the atmosphere in which they work and take necessary steps to minimize the risks. Careful practice avoids many accidents in mortuary.

The present study concludes that testing of HBV in medico legal autopsies is a convenient and effective method in monitoring the surveillance of HBV-infection in the general population and it can be used for epidemiological studies. It could be used along with unlinked anonymous tests from hospital and other similar patient materials.

Testing for HBV may also be desired for safety reasons in mortuaries[81]. In screening postmortem blood for HbSAg, the present study represents that HbSAg ELISA kit is simple, rapid, no special equipment are required, even whole blood can be used and has very high sensitivity of 99.58% and specificity of 100% and

Out of 515 subjects in this study group, 18 cases were positive for HBV and all were previously unknown seropositive cases. Though the rate of infection appears to be less than 1%, even with a needle stick, it is not practical or economical to take universal precautions with every autopsy. It would, therefore, be advantageous to know a deceased HBV serological status prior to autopsy. Screening may be worthwhile in cases at high risk of HBV infection where the HBV status is not known at the time of postmortem.

UNIVERSAL PRECAUTIONS WHICH SHOULD BE FOLLOWED DURING ROUTINE AUTOPSY PROCEDURES

Universal precautions should be followed for blood, vaginal secretions and semen as well as to peritoneal, pericardial cerebrospinal, synovial, pleura fluids and it is believed that tears, nasal secretions, sweat, sputum, urine, faeces and vomitus and amniotic fluids are not infectious unless they contain visible blood.

Only experts and health care professionals those who are skilled in handling the infected materials are allowed to enter in to the postmortem examination room. Only those experienced professionals should conduct the autopsy examination as it is proved through many studies that the risk of accidental infection was more common, when the post mortem procedure was done by an inexperienced person. Studies reported that the occurrence of lacerated wound in the body of the performer in 1 in every 11 autopsies conducted by inexperienced persons. Postmortem examination should not involve those persons with breach in the skin or mucosa or body immunity.

The internal atmosphere of the postmortem examination room should be in such a way with sufficient space to avoid overcrowding and provide proper ventilation. Protective measures such as wearing gloves, headwear, masks, eyewear, shoes and full-covered gown should be given to and used by the mortuary staff while they are doing the postmortem work. Gloves should be checked often for cuts and puncture to prevent them from getting contaminated and infected by infectious leakage. Wearing double gloves during the procedure will help us to minimize the risk of cutaneous exposure to the infected blood

It should become a routine practice of washing your hands, after handling contaminated and infected blood and body fluids and also after

the removal of gloves and check for the perforation in the removed gloves. Gloves should be washed as routine procedure after autopsy to remove the contaminated blood and/or other body fluids, as there is high chance of transferring the contamination to all the surfaces coming in contact with the performers. Sharp instruments should not transferred directly from one’s hand to other's hand while doing the postmortem work to other, without a vehicle in between All Disposable sharp materials should be carefully placed in puncture resistant containers and later disposed in a very safe manner.

As prevention is always better than cure, the following preventive measures such as screening and immunization of the persons who are considered as high-risk groups based on the history of exposure to infection and infected personnel, risk involved in their routine day to day practices and risk involved in the occupation in which they are engaged. Recommended safety measures should be followed in the area of Child Birth, Blood Transfusion, Injections, Sex and in all places wherever we comes in contact with blood and body fluids.

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DOI: 10.4323/RJLM.2011.183 © 2011 ROMANIAN SOCIETY OF LEGAL MEDICINE

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ANNEXURE

STUDY OF SEROPREVALENCE OF HEPATITIS B VIRUS IN MEDICO LEGAL AUTOPSIES

Proforma:

Postmortem No: Date: Age: Sex:

History of the case and Manner of Death:

Marital status:

Occupation:

Brought Dead or Hospital treated:

Date and Time of Death:

Date and Time of Postmortem examination:

Known case of Hepatitis B: yes/No

Samples Collected:

1) Blood from left ventricle: Yes/No

Investigation: To detect the presence of HBsAg in the collected sample using Human HBsAg ELISA Kit.

MASTER CHART

Serial No	P.M.No	Age	Sex	Marital Status	Manner of Death	Occupation	Brought Dead or Treated	Date & Time of Death	Date and Time of Autopsy	*Time since death in Hours*					*Known HBV status*	Sample Result - HBsAg Positive
										< 6	*6 – 12*	*12 – 24*	*24 - 36*	*>36*		
1.	867/14	60	M	Married	Hanging	Farmer	Treated	14.05.14 07.25 A.M	14.05.14 01.00.P.M	Yes					*No*	*No*
2.	868/14	44	M	Married	RTA	Farmer	Brought dead	13.05.14 12.30P.M.	14.05.14 01.30 P.M				*Yes*		*No*	*No*
3.	869/14	54	M	Married	RTA	Coolie	Treated	13.05.14 10.15 A.M	14.05.14 02.00P.M.				*Yes*		*No*	*No*
4.	870/14	50	M	Married	RTA	Coolie	Treated	12.05.14 04.45 P.M	14.05.14 02.00 P.M					*Yes*	*No*	*No*
5.	871/14	58	M	Married	Fall	Coolie	Treated	14.05.14 04.00 A.M	14.05.14 02.00 P.M		*Yes*				*No*	*No*
6.	872/14	20	M	Married	Hanging	Coolie	Brought dead	14.05.14 1.20 A.M	15.05.14 12.10 P.M				*Yes*		*No*	*No*
7.	873/14	30	F	Married	Natural	House wife	Brought dead	14.05.14 02.00 A.M	15.05.14 01.00 P.M				*Yes*		*No*	*No*
8.	874/14	27	M	Married	RTA	Coolie	Treated	15.05.14 05.30 A.M	15.05.14 02.00 P.M		*Yes*				*No*	*No*
9.	876/14	48	M	Married	Fall	Private	Treated	15.05.14 06.20 A.M	15.05.14 04.00 P.M		*Yes*				*No*	*No*
10.	877/14	25	M	Single	TTA	Cook	Brought dead	15.05.14 07.40 A.M	15.05.14 05.00 P.M		*Yes*				*No*	*No*
11.	878/14	23	M	Single	RTA	Driver	Treated	15.05.14 01.00 P.M	16.05.14 11.00 A.M			*Yes*			*No*	Yes
12.	879/14	28	M	Married	RTA	Driver	Treated	16.05.14 04.50 A.M	16.05.14 11.55 A.M		*Yes*				*No*	*No*
13.	881/14	65	M	Married	RTA	Coolie	Treated	15.05.14 06.15 A.M	16.05.14 01.15 P.M				*Yes*		*No*	*No*
14.	882/14	24	M	Single	RTA	Coolie	Treated	15.05.14 01.30 P.M	16.05.14 04.00 P.M				*Yes*		*No*	*No*
15.	883/14	32	M	Married	Fall	Coolie	Treated	16.05.14 07.35 A.M	17.05.14 11.00 A.M				*Yes*		*No*	*No*
16.	885/14	49	M	Married	RTA	Mason	Brought	16.05.14	17.05.14			*Yes*			*No*	*No*

							dead	02.30 P.M	12.40 P.M							
17.	886/14	21	M	Single	RTA	Private	Treated	17.05.14 03.30 P.M	18.05.14 12.15 P.M			*Yes*			*No*	*No*
18.	887/14	29	M	Married	RTA	Coolie	Brought dead	17.05.14 06.30 P.M	18.05.14 01.00 P.M			*Yes*			*No*	*No*
19.	888/14	90	M	Married	Hanging	Coolie	Treated	18.05.14 12.30 A.M	18.05.14 01.15 P.M			*Yes*			*No*	*No*
20.	889/14	29	M	Married	Snake Bite	Coolie	Treated	18.05.14 07.50 A.M	19.05.14 10.15 A.M				*Yes*		*No*	*No*
21.	890/14	27	M	Married	RTA	Coolie	Treated	18.05.14 04.02 P.M	19.05.14 12.40 P.M			*Yes*			*No*	*No*
22.	891/14	40	M	Married	RTA	Coolie	Treated	18.05.14 06.45 P.M	19.05.14 01.30 P.M			*Yes*			*No*	*No*
23.	892/14	58	F	Married	RTA	Coolie	Brought dead	18.05.14 07.30 P.M	19.05.14 02.30 P.M			*Yes*			*No*	*No*
24.	893/14	15	M	Single	RTA	Student	Treated	19.05.14 07.15 A.M	19.05.14 03.30 P.M		*Yes*				*No*	*No*
25.	895/14	43	M	Married	RTA	Farmer	Treated	19.05.14 06.00 A.M	19.05.14 04.00 P.M		*Yes*				*No*	*No*
26.	896/14	27	M	Married	TTA	Private	Brought dead	19.05.14 12.05 P.M	20.05.14 10.30 A.M			*Yes*			*No*	***Yes***
27.	897/14	70	M	Married	Fall	Merchant	Treated	19.05.14 07.15 P.M	20.05.14 11.30 A.M			*Yes*			*No*	*No*
28.	898/14	25	M	Single	RTA	Painter	Treated	19.05.14 07.10 P.M	20.05.14 12.15 P.M			*Yes*			*No*	*No*
29.	899/14	65	M	NK	Natural	NK	Brought dead	19.05.14 11.40 A.M	20.05.14 12.40 P.M				*Yes*		*No*	*No*
30.	900/14	26	M	Married	RTA	Coolie	Brought dead	19.05.14 10.45 P.M	20.05.14 12.30 P.M			*Yes*			*No*	*No*
31.	901/14	43	M	Married	Natural	Coolie	Brought dead	20.05.14 09.05 A.M	20.05.14 01.30 P.M	Yes					*No*	*No*
32.	902/14	28	F	Married	Snake Bite	Coolie	Treated	19.05.14 08.30 P.M	20.05.14 01.30 P.M			*Yes*			*No*	*No*
33.	903/14	45	M	Married	Fall	Painter	Treated	19.05.14 11.15 P.M	20.05.14 02.15 P.M			*Yes*			*No*	*No*
34.	905/14	28	F	Married	TTA	Coolie	Treated	19.05.14	21.05.14					*Yes*	*No*	*No*

								03.45 P.M	01.30 P.M							
35.	906/14	19	M	Single	Snake Bite	Coolie	Treated	20.05.14 10.30 P.M	21.05.14 01.30 P.M			*Yes*			*No*	*No*
36.	907/14	21	M	Single	RTA	Business	Brought dead	21.05.14 07.30 A.M	21.05.14 01.40 P.M		*Yes*				*No*	*No*
37.	909/14	63	M	Married	RTA	Coolie	Treated	21.05.14 01.15 A.M	21.05.14 04.00 P.M			*Yes*			*No*	*No*
38.	910/14	44	M	Married	TTA	Govt	Brought dead	21.05.14 11.55 A.M	22.05.14 11.00 A.M			*Yes*			*No*	*No*
39.	913/14	45	M	Married	Natural	Coolie	Brought dead	22.05.14 10.45 A.M	22.05.14 03.30 P.M	Yes					*No*	*No*
40.	915/14	38	M	Married	Hanging	Tailor	Treated	22.05.14 05.30 P.M	23.05.14 12 Noon			*Yes*			*No*	*No*
41.	916/14	57	M	Married	Poisoning	Tailor	Treated	22.05.14 05.00 P.M	23.05.14 12.45 P.M			*Yes*			*No*	*No*
42.	917/14	20	M	Single	RTA	Student	Treated	23.05.14 03.50 A.M	23.05.14 02.15 P.M		*Yes*				*No*	*No*
43.	918/14	42	M	Married	RTA	Driver	Treated	23.05.14 07.40 A.M	23.05.14 03.15 P.M		*Yes*				*No*	*No*
44.	919/14	60	M	Married	Murder	Coolie	Treated	23.05.14 05.30 A.M	23.05.14 03.30 P.M		*Yes*				*No*	*No*
45.	920/14	40	M	Married	Hanging	Coolie	Brought dead	23.05.14 07.45 P.M	24.05.14 11.00 A.M			*Yes*			*No*	***Yes***
46.	921/14	23	M	Single	Fall	Coolie	Treated	22.05.14 08.45 P.M	24.05.14 11.15 A.M					*Yes*	*No*	*No*
47.	922/14	62	M	Married	RTA	Private	Treated	23.05.14 11.00 P.M	24.05.14 01.00 P.M			*Yes*			*No*	*No*
48.	923/14	45	F	Married	RTA	Tailor	Treated	23.05.14 02.05 P.M	24.05.14 12 Noon		*Yes*				*No*	*No*
49.	926/14	20	M	Single	Fall	Painter	Treated	23.05.14 05.10 P.M	24.05.14 03.00 P.M			*Yes*			*No*	*No*
50.	927/14	53	M	Married	Fall	Painter	Treated	24.05.14 07.00 A.M	25.05.14 10.30 A.M				*Yes*		*No*	*No*
51.	928/14	27	M	Married	RTA	Private	Treated	24.05.14 11.25 P.M	25.05.14 11.45 A.M			*Yes*			*No*	*No*
52.	931/14	27	F	Married	Poisoning	Coolie	Treated	23.05.14	25.05.14					*Yes*	*No*	*No*

								11.00 P.M	02.15 P.M							
53.	932/14	65	M	Married	Fall	Coolie	Treated	25.05.14 06.00 A.M	25.05.14 03.30 P.M		*Yes*				*No*	*No*
54.	933/14	45	F	Married	TTA	House wife	Brought dead	25.05.14 11.20 A.M	25.05.14 03.45 P.M	Yes					*No*	*No*
55.	934/14	48	F	Married	Fall	Coolie	Treated	24.05.14 10.50 P.M	25.05.14 4.15 P.M			*Yes*			*No*	*No*
56.	935/14	26	F	Married	Poisoning	Farmer	Treated	25.05.14 01.45 P.M	26.05.14 10.15 A.M			*Yes*			*No*	*No*
57.	937/14	25	M	Single	Murder	Coolie	Treated	25.05.14 04.35 P.M	26.05.14 11.30 A.M			*Yes*			*No*	*No*
58.	938/14	49	F	Married	Poisoning	House wife	Treated	25.05.14 02.45 P.M	26.05.14 12.15 P.M			*Yes*			*No*	*No*
59.	939/14	29	M	Single	RTA	Coolie	Treated	25.05.14 11.00 P.M	26.05.14 12.40 P.M			*Yes*			*No*	*No*
60.	940/14	62	M	Married	RTA	Security	Treated	25.05.14 09.00 A.M	26.05.14 01.45 P.M				*Yes*		*No*	*No*
61.	941/14	45	M	Married	RTA	Coolie	Treated	25.05.14 09.30P.M.	26.05.14 04.00 P.M			*Yes*			*No*	*No*
62.	942/14	37	M	Married	RTA	Driver	Treated	26.05.14 11.30 A.M	27.05.14 10.15 A.M			*Yes*			*No*	*No*
63.	943/14	45	M	Married	RTA	Merchant	Treated	26.05.14 04.30 P.M	27.05.14 12.05 P.M			*Yes*			*No*	*No*
64.	947/14	58	M	Married	Fall	Security	Brought dead	27.05.14 05.10 A.M	27.05.14 01.40 P.M		*Yes*				*No*	***Yes***
65.	948/14	28	M	Married	Accident	Coolie	Treated	27.05.14 05.30 A.M	27.05.14 04.00 P.M		*Yes*				*No*	*No*
66.	949/14	24	M	Single	Poisoning	Student	Treated	27.05.14 09.00 A.M	28.05.14 10.45 A.M				*Yes*		*No*	*No*
67.	950/14	18	M	Single	TTA	Coolie	Treated	28.05.14 08.10 A.M	28.05.14 11.00 A.M	Yes					*No*	*No*
68.	951/14	57	M	Married	RTA	Govt	Treated	27.05.14 02.45 P.M	28.05.14 11.30 A.M			*Yes*			*No*	*No*
69.	952/14	65	M	Married	Fall	Coolie	Treated	27.05.14 07.30 P.M	28.05.14 02.30 P.M			*Yes*			*No*	*No*
70.	953/14	55	M	Married	RTA	Coolie	Treated	28.05.14	28.05.14		*Yes*				*No*	*No*

								04.15 A.M	03.45 P.M							
71.	958/14	87	M	Married	RTA	Retd	Treated	28.05.14 07.00 P.M	29.07.14 12.15 P.M					*Yes*	*No*	*No*
72.	960/14	40	M	Married	RTA	Coolie	Treated	28.05.14 07.30 P.M	29.05.14 02.15 P.M			*Yes*			*No*	*No*
73.	961/14	18	M	Single	Electrocut ion	Student	Treated	29.05.14 07.20 P.M	30.05.14 11.40 A.M			*Yes*			*No*	*No*
74.	962/14	65	M	Married	Fall	Govt	Treated	30.05.14 04.10 A.M	30.05.14 01.00 P.M		*Yes*				*No*	*No*
75.	963/14	30	M	Married	RTA	Coolie	Treated	30.05.14 04.30 A.M	30.05.14 01.30 P.M		*Yes*				*No*	*No*
76.	964/14	39	M	Married	Murder	Tailor	Treated	29.05.14 04.00 P.M	30.05.14 02.00 P.M			*Yes*			*No*	*No*
77.	967/14	22	F	Married	Poisoning	House wife	Treated	29.05.14 07.40 P.M	30.05.14 04.30 P.M			*Yes*			*No*	*No*
78.	970/14	57	F	Married	Poisoning	House wife	Treated	30.05.14 08.25 P.M	31.05.14 01.40 P.M			*Yes*			*No*	***Yes***
79.	971/14	55	F	Married	Snake Bite	House wife	Treated	31.05.14 05.30 A.M	31.05.14 03.15 P.M		*Yes*				*No*	*No*
80.	973/14	43	M	Married	RTA	Driver	Treated	31.05.14 03.00 P.M	01.06.14 10.15 A.M			*Yes*			*No*	*No*
81.	974/14	37	M	Single	Fall	Painter	Treated	31.05.14 03.50 P.M	01.06.14 11.15 A.M			*Yes*			*No*	*No*
82.	975/14	16	M	Single	Poisoning	Student	Treated	31.05.14 09.30 P.M	01.06.14 11.40 A.M			*Yes*			*No*	*No*
83.	976/14	65	M	Married	Natural	Coolie	Brought dead	30.05.14 09.10 P.M	01.06.14 12.15 P.M					*Yes*	*No*	*No*
84.	977/14	45	M	Married	RTA	Coolie	Brought dead	31.05.14 09.08 A.M	01.06.14 01.00 P.M				*Yes*		*No*	*No*
85.	978/14	22	M	Single	RTA	Coolie	Treated	31.05.14 05.40 P.M	01.06.14 01.30 P.M			*Yes*			*No*	*No*
86.	979/14	70	F	Married	Natural	House wife	Brought dead	31.05.14 03.40P.M.	01.06.14 04.00 P.M			*Yes*			*No*	*No*
87.	980/14	27	M	Married	Poisoning	Coolie	Treated	01.06.14 04.30 A.M	01.06.14 03.00 P.M		*Yes*				*No*	*No*
88.	981/14	30	M	Married	RTA	Coolie	Brought	01.06.14	02.06.14			*Yes*			*No*	*No*

							dead	08.20 P.M	12.15 P.M							
89.	982/14	60	F	Married	RTA	Coolie	Brought dead	01.06.14 01.30 P.M	02.06.14 01.15 P.M			*Yes*			*No*	*No*
90.	983/14	35	M	Single	RTA	Coolie	Brought dead	01.06.14 03.00 A.M	02.06.14 02.00 P.M				*Yes*		*No*	*No*
91.	985/14	34	M	Married	Natural	Coolie	Brought dead	02.06.14 10.45 A.M	03.06.14 11.15 A.M				*Yes*		*No*	*No*
92.	986/14	38	M	Married	RTA	Business	Treated	02.06.14 09.40 A.M	03.06.14 11.40 A.M				*Yes*		*No*	*No*
93.	987/14	22	M	Single	RTA	Coolie	Treated	02.06.14 10.35 P.M	03.06.14 01.00 P.M			*Yes*			*No*	*No*
94.	988/14	45	M	Married	RTA	Coolie	Treated	01.06.14 01.15 P.M	03.06.14 01.40 P.M					Yes	*No*	*No*
95.	989/14	22	M	Single	RTA	Driver	Brought dead	03.06.14 04.45 P.M	03.06.14 03.30 P.M			*Yes*			*No*	***Yes***
96.	990/14	45	F	Married	RTA	House wife	Treated	03.06.14 07.25 A.M	03.06.14 04.15 P.M		*Yes*				*No*	*No*
97.	991/14	26	M	Single	RTA	Driver	Treated	03.06.14 03.45 P.M	04.06.14 12.Noon			*Yes*			*No*	*No*
98.	992/14	40	M	Married	RTA	Driver	Treated	03.06.14 09.00 P.M	04.06.14 12.30 P.M			*Yes*			*No*	*No*
99.	993/14	60	M	Married	TTA	Retd	Brought dead	03.06.14 07.00 P.M	04.06.14 01.30 P.M			*Yes*			*No*	*No*
100.	995/14	50	M	Married	Fall	Coolie	Treated	04.06.14 02.35 A.M	04.06.14 03.30 P.M			*Yes*			*No*	*No*
101.	998/14	45	M	Married	RTA	Coolie	Brought dead	04.06.14 02.25 P.M	05.06.14 11.15 A.M			*Yes*			*No*	*No*
102.	999/14	42	M	Married	TTA	Coolie	Brought dead	04.06.14 06.05 P.M	05.06.14 11.40 A.M			*Yes*			*No*	*No*
103.	1000/14	44	M	Married	RTA	Security	Treated	04.06.14 11.00 P.M	05.06.14 12.Noon			*Yes*			*No*	*No*
104.	1001/14	65	M	Married	RTA	Coolie	Treated	04.06.14 08.15 P.M	05.06.14 01.30 P.M			*Yes*			*No*	*No*
105.	1002/14	27	M	Married	Fall	Cleaner	Treated	04.06.14 11.00 A.M	05.06.14 02.00 P.M				*Yes*		*No*	*No*
106.	1003/14	21	M	Single	RTA	Painter	Treated	04.06.14	05.06.14			*Yes*			*No*	*No*

								11.00 P.M	02.00 P.M							
107.	1004/14	68	M	Married	RTA	Private	Treated	05.06.14 06.55 A.M	05.06.14 02.30 P.M		*Yes*				*No*	*No*
108.	1005/14	30	M	Married	RTA	Coolie	Treated	04.06.14 11.00 A.M	05.06.14 02.15 P.M				*Yes*		*No*	*No*
109.	1007/14	22	M	Single	TTA	Coolie	Treated	05.06.14 01.20 A.M	05.06.14 03.20 P.M			*Yes*			*No*	*No*
110.	1009/14	28	M	Married	RTA	Driver	Treated	04.06.14 10.25 P.M	05.06.14 04.00 P.M			*Yes*			*No*	*No*
111.	1010/14	20	M	Single	RTA	Student	Treated	05.06.14 12.00P.M.	06.06.14 04.15 P.M				*Yes*		*No*	*No*
112.	1011/14	25	M	Married	RTA	Coolie	Treated	05.06.14 05.45 A.M	05.06.14 04.20 P.M		*Yes*				*No*	*No*
113.	1013/14	50	M	Married	RTA	Security	Treated	05.06.14 12.40 A.M	06.06.14 11,40 A.M				*Yes*		*No*	*No*
114.	1014/14	38	M	Married	RTA	Driver	Treated	05.06.14 05.30 P.M	06.06.14 12 Noon			*Yes*			*No*	*No*
115.	1015/14	41	M	Married	RTA	Driver	Brought dead	05.06.14 01.10 P.M	06.06.14 12.10 P.M			*Yes*			*No*	*No*
116.	1016/14	23	M	Single	RTA	Private	Treated	06.06.14 02.40 A.M	06.06.14 12.30 P.M		*Yes*				*No*	*No*
117.	1019/14	36	M	Married	Poisoning	Coolie	Treated	05.06.14 10.30 P.M	06.06.14 01.30 P.M			*Yes*			*No*	*No*
118.	1020/14	30	M	Married	RTA	Fish man	Treated	05.06.14 09.00 P.M	06.06.14 01.40 P.M			*Yes*			*No*	*No*
119.	1021/14	19	M	Single	Natural	Coolie	Treated	05.06.14 11.25 P.M	06.06.14 02.00 P.M			*Yes*			*No*	*No*
120.	1022/14	52	M	Married	Fall	Driver	Treated	05.06.14 04.30 P.M	06.06.14 04.15 P.M			*Yes*			*No*	*No*
121.	1023/14	22	M	Single	TTA	Coolie	Brought dead	07.06.14 07.15 A.M	07.06.14 10.45 A.M	Yes					*No*	*No*

122.	1025/14	45	M	Married	Fall	Business	Treated	06.06.14 04.00 A.M	07.06.14 12.30 P.M				*Yes*		*No*	*No*
123.	1026/14	55	M	Married	Natural	Coolie	Brought dead	06.06.14 05.10 P.M	07.06.14 12.30 P.M			*Yes*			*No*	*No*
124.	1028/14	55	M	NK	Natural	NK	Brought dead	30.05.14 11.45 A.M	07.06.14 01.30 P.M					*Yes*	*No*	*NO*
125.	1032/14	45	M	Married	RTA	Private	Treated	06.06.14 09.30 P.M	07.06.14 02.15 P.M			*Yes*			*No*	*No*
126.	1034/14	55	F	Married	RTA	Coolie	Treated	06.06.14 04.00P.M	07.06.14 02.45P.M			*Yes*			*No*	*No*
127.	1035/14	27	M	Married	Electrocut ion	Coolie	Treated	06.06.14 08.20 P.M	07.06.14 03.15 P.M			*Yes*			*No*	*No*
128.	1036/14	60	M	Married	Natural	Driver	Brought dead	06.06.14 09.10 P.M	07.06.14 03.45 P.M			Yes			*No*	*No*
129.	1038/14	23	M	Married	RTA	Driver	Treated	07.06.14 12.30 P.M	08.06.14 11.30 A.M			*Yes*			*No*	*No*
130.	1039/14	35	F	Married	RTA	Coolie	Treated	07.06.14 09.45 P.M	08.06.14 11.40 A.M			*Yes*			*No*	*No*
131.	1040/14	38	F	NK	Murder	NK	Treated	07.06.14 07.00 A.M	08.06.14 11.40 A.M				*Yes*		*No*	*No*
132.	1041/14	63	M	Married	Murder	Private	Treated	08.06.14 12.15 A.M	08.06.14 12.40 P.M			*Yes*			*No*	*No*
133.	1042/14	20	M	Single	RTA	Coolie	Treated	08.06.14 07.40 A.M	08.06.14 01.30 P.M	Yes					*No*	*No*
134.	1043/14	50	M	Married	Poisoning	Coolie	Treated	08.06.14 02.20 A.M	08.06.14 01.30 P.M		Yes				*No*	*No*
135.	1044/14	33	M	Married	TTA	Coolie	Brought dead	08.06.14 10.30 A.M	08.06.14 02.00 P.M	Yes					*No*	*No*
136.	1045/14	42	M	Married	RTA	Coolie	Treated	08.06.14 12.15 A.M	08.06.14 02.30 P.M			Yes			*No*	*No*
137.	1046/14	50	F	Married	RTA	House wife	Treated	08.06.14 07.50 A.M	09.06.14 11.30 A.M				*Yes*		*No*	*No*
138.	1047/14	50	M	Married	RTA	Farmer	Treated	08.06.14 01.15 P.M	09.06.14 11.30 A.M			*Yes*			*No*	*No*
139.	1050/14	53	M	Married	TTA	Security	Brought dead	09.06.14 11.10 A.M	09.06.14 03.00 P.M	Yes					*No*	*No*

140.	1051/14	35	M	Married	RTA	Coolie	Treated	09.06.14 02.25 A.M	10.06.14 12.00 P.M				*Yes*		*No*	*No*
141.	1052/14	60	M	Married	RTA	Farmer	Treated	09.06.14 11.25 A.M	10.06.14 12.30 P.M				*Yes*		*No*	*No*
142.	1053/14	32	M	Married	Murder	Coolie	Treated	09.06.14 01.10 A.M	10.06.14 12.30 P.M			*Yes*			*No*	*No*
143.	1055/14	43	M	Married	Natural	Driver	Treated	09.06.14 10.45 A.M	10.06.14 01.30 P.M				*Yes*		*No*	*No*
144.	1058/14	45	M	Married	Fall	Coolie	Treated	10.06.14 09.30 A.M	10.06.14 04.00 P.M		*Yes*				*No*	*No*
145.	1059/14	21	M	Married	RTA	Coolie	Treated	09.06.14 04.30 P.M	10.06.14 04.15 P.M			*Yes*			*No*	*No*
146.	1060/14	45	M	Married	Poisoning	Driver	Treated	10.06.14 12.15 A.M	10.06.14 04.30 P.M			*Yes*			*No*	*No*
147.	1061/14	35	F	Married	Poisoning	Coolie	Brought dead	10.06.14 12.30 A.M	10.06.14 04.45 P.M			*Yes*			*No*	***Yes***
148.	1063/14	62	M	Married	RTA	Retd	Treated	10.06.14 08.45 P.M	11.06.14 11.30 A.M			Yes			*No*	*No*
149.	1064/14	51	M	Married	RTA	Coolie	Treated	11.06.14 01.45 A.M	11.06.14 11.40 A.M		Yes				*No*	*No*
150.	1067/14	60	M	Married	RTA	Coolie	Treated	11.06.14 01.00 A.M	11.06.14 01.15 P.M			Yes			*No*	*No*
151.	1068/14	45	M	Married	RTA	Driver	Treated	10.06.14 08.20 P.M	11.06.14 03.30 P.M			Yes			*No*	*No*
152.	1069/14	43	M	Married	Natural	Driver	Brought dead	11.06.14 12 Noon	12.06.14 11.00 A.M			Yes			*No*	***Yes***
153.	1071/14	19	M	Single	RTA	Coolie	Treated	11.06.14 01.30 P.M	12.06.14 11.30 A.M			Yes			*No*	*No*
154.	1072/14	45	M	Married	RTA	Coolie	Treated	11.06.14 10.00 A.M	12.06.14 12 Noon				Yes		*No*	*No*
155.	1074/14	58	M	Married	RTA	Coolie	Treated	11.06.14 01.00 P.M	12.06.14 01.30 P.M				Yes		*No*	*No*
156.	1075/14	24	M	Married	RTA	Coolie	Brought dead	12.06.14 09.25 A.M	12.06.14 01.30 P.M		Yes				*No*	*No*
157.	1079/14	43	M	Married	TTA	Coolie	Treated	12.06.14 04.00 A.M	12.06.14 03.30 P.M		Yes				*No*	*No*

158.	1080/14	65	M	Married	Natural	Retd	Brought dead	12.06.14 09.00 A.M	13.06.14 11.30 A.M				Yes		*No*	*No*
159.	1081/14	58	M	Married	Fall	Coolie	Treated	12.06.14 01.05 P.M	13.06.14 11.00 A.M			*Yes*			*No*	*No*
160.	1082/14	23	M	Single	RTA	Private	Treated	12.06.14 02.00P.M	13.06.14 12 Noon			*Yes*			*No*	*No*
161.	1083/14	42	M	Married	Electrocution	Coolie	Brought dead	12.06.14 03.15 P.M	13.06.14 12 Noon			*Yes*			*No*	*No*
162.	1084/14	53	M	Married	TTA	Govt	Brought dead	13.06.14 01.45 P.M	13.06.14 02.45 P.M	Yes					*No*	*No*
163.	1085/14	50	M	Married	Murder	Private	Treated	12.06.14 11.40 A.M	13.06.14 03.00 P.M				*Yes*		*No*	*No*
164.	1088/14	65	M	Married	Fall	Coolie	Treated	13.06.14 12.35 P.M	14 06.14 11.00 A.M			*Yes*			*No*	*No*
165.	1090/14	65	M	Married	Poisoning	Coolie	Treated	13.06.14 01.40 P.M	14.06.14 12 Noon			*Yes*			*No*	*No*
166.	1091/14	25	M	Married	RTA	Driver	Treated	14.06.14 02.30 A.M	14.06.14 12.30 P.M		*Yes*				*No*	*No*
167.	1093/14	35	M	Married	Natural	Coolie	Brought dead	13.06.14 06.00 P.M	14.06.14 02.30 P.M			*Yes*			*No*	*No*
168.	1095/14	45	M	Married	RTA	Coolie	Treated	14.06.14 06.10 A.M	14.06.14 03.30 P.M		Yes				*No*	*No*
169.	1096/14	18	M	Single	TTA	Student	Brought dead	14.06.14 09.05 P.M	15.06.14 10.45A.M			Yes			*No*	*No*
170.	1098/14	28	M	Married	RTA	Carpenter	Treated	14.06.14 06.10 P.M	15.06.14 01.00 P.M			Yes			*No*	*No*
171.	1101/14	48	M	Married	RTA	Coolie	Brought dead	15.06.14 09.50 A.M	15.06.14 03.40 P.M	Yes					*No*	*No*
172.	1103/14	44	M	Married	RTA	Mason	Treated	15.06.14 12.18 P.M	16.06.14 12.35 P.M				Yes		*No*	*No*
173.	1104/14	60	M	Married	Fall	Security	Treated	16.06.14 12.10.A.M	16.06.14 12.55 P.M			*Yes*			*No*	*No*
174.	1105/14	18	M	Single	RTA	Coolie	Treated	15.06.14 08.00 P.M	16.06.14 01.30 P.M			*Yes*			*No*	*No*
175.	1106/14	27	Transg	Single	Hanging	Coolie	Treated	15.06.14 08.50 P.M	16.06.14 01.30 P.M			*Yes*			*No*	***Yes***

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176.	1107/14	80	M	Married	RTA	Farmer	Treated	16.06.14 05.30 A.M	16.06.14 02.10 P.M		Yes				*No*	*No*
177.	1109/14	48	M	Married	Natural	Govt	Brought dead	14.06.14 11.00 A.M	16.06.14 02.35 P.M					Yes	*No*	*No*
178.	1111/14	29	M	Married	RTA	Coolie	Treated	16.06.14 08.10 A.M	17.06.14 11.10 A.M				Yes		*No*	*No*
179.	1112/14	32	M	Married	Hanging	Driver	Treated	16.06.14 05.00 A.M	17.06.14 12.10 P.M				*Yes*		*No*	*No*
180.	1113/14	45	M	Married	RTA	Coolie	Treated	16.06.14 07.25 A.M	17.06.14 12.35 P.M				Yes		*No*	*No*
181.	1114/14	30	M	Married	Fall	Coolie	Treated	16.06.14 05.05 P.M	17.06.14 01.00 P.M			*Yes*			*No*	*No*
182.	1115/14	25	F	Married	Poisoning	House wife	Treated	16.06.14 04.00 P.M	17.06.14 03.30 P.M			*Yes*			*No*	*No*
183.	1116/14	30	M	Married	RTA	Goldsmith	Treated	16.06.14 11.10 P.M	17.06.14 03.40 P.M			*Yes*			*No*	*No*
184.	1118/14	37	M	Married	RTA	Electrician	Treated	17.07.14 12.30 P.M	18.06.14 10.30 A.M			*Yes*			*No*	*No*
185.	1119/14	23	M	Married	RTA	Coolie	Treated	17.06.14 03.50 P.M	18.06.14 12.40 P.M			*Yes*			*No*	*No*
186.	1120/14	25	M	Married	Suspicious death	Driver	Brought dead	17.06.14 07.00 A.M	18.06.14 12.55 P.M				Yes		*No*	*No*
187.	1121/14	76	M	Married	RTA	Farmer	Treated	17.06.14 11.45 A.M	18.06.14 02.50 P.M				Yes		*No*	*No*
188.	1123/14	32	M	Married	RTA	Electrician	Treated	18.06.14 02.20 A.M	18.06.14 03.50 P.M			*Yes*			*No*	*No*
189.	1124/14	60	F	Married	TTA	Coolie	Treated	18.06.14 08.30 A.M	18.06.14 03.50 P.M		*Yes*				*No*	*No*
190.	1125/14	41	M	Married	RTA	Coolie	Treated	18.06.14 06.35 A.M	19.06.14 10.40 A.M				*Yes*		*No*	*No*
191.	1126/14	54	M	Married	Fall	Cine Field	Treated	18.06.14 08.15 P.M	19.06.14 12.45 P.M			*Yes*			*No*	*No*
192.	1127/14	50	M	Married	RTA	Driver	Treated	19.06.14 07.10 A.M	19.06.14 02.40 P.M		*Yes*				*No*	*No*

193.	1128/14	50	M	Married	RTA	Coolie	Treated	19.06.14 02.00 A.M	19.06.14 03.10 P.M			*Yes*			*No*	*No*
194.	1130/14	64	M	Married	RTA	Mechanic	Treated	19.06.14 10.40 A.M	20.06.14 11.00 A.M				*Yes*		*No*	*No*
195.	1131/14	24	F	Married	Poisoning	House wife	Treated	18.06.14 02.15 A.M	20.06.14 11.30 A.M					*Yes*	*No*	*No*
196.	1132/14	32	M	Married	RTA	Coolie	Treated	19.06.14 04.00 P.M	20.06.14 12.15 P.M			*Yes*			*No*	*No*
197.	1133/14	13	M	Single	RTA	Student	Brought dead	19.06.14 04.35 P.M	20.06.14 12.10 P.M			Yes			*No*	*No*
198.	1134/14	25	M	Single	RTA	Private	Treated	19.06.14 06.05 A.M	20.06.14 12.40 P.M				Yes		*No*	*No*
199.	1135/14	55	M	Married	Natural	Coolie	Brought dead	19.06.14 12.35 P.M	20.06.14 01.10 P.M				*Yes*		*No*	*No*
200.	1136/14	50	M	Married	RTA	Private	Treated	20.06.14 01.30 A.M	20.06.14 01.10 P.M		*Yes*				*No*	*No*
201.	1137/14	62	M	Married	RTA	Security	Treated	20.06.14 12.35 A.M	20.06.14 01.40 P.M			*Yes*			*No*	*No*
202.	1138/14	34	M	Married	RTA	Coolie	Treated	19.06.14 09.45 A.M	20.06.14 01.30 P.M				*Yes*		*No*	*No*
203.	1146/14	58	M	Married	RTA	Coolie	Treated	21.06.14 01.30 A.M	21.06.14 12.10P.M		Yes				*No*	*No*
204.	1148/14	45	M	Married	RTA	Farmer	Treated	21.06.14 04.30 A.M	21.06.14 02.40 P.M		Yes				*No*	*No*
205.	1150/14	54	M	Married	RTA	Tailor	Brought dead	21.06.14 12.40 P.M	22.06.14 10.30 A.M			*Yes*			*No*	*No*
206.	1151/14	40	M	Married	RTA	Painter	Treated	21.06.14 02.35 P.M	22.06.14 12.45 P.M			*Yes*			*No*	*No*
207.	1152/14	35	M	Married	Poisoning	Coolie	Treated	21.06.14 09.15 P.M	22.06.14 02.30 P.M			*Yes*			*No*	*No*
208.	1153/14	40	M	Married	RTA	Driver	Treated	21.06.14 11.30 P.M	22.06.14 03.30 P.M			*Yes*			*No*	*No*
209.	1154/14	55	F	Married	Suspicious Death	Coolie	Treated	22.06.14 10.20 A.M	23.06.14 10.40 A.M				Yes		*No*	*No*
210.	1155/14	35	M	Married	RTA	Coolie	Treated	22.06.14 05.30 P.M	23.06.14 11.10 A.M			*Yes*			*No*	*No*

211.	1156/14	45	F	Married	RTA	House wife	Treated	22.06.14 02.30 P.M	23.06.14 11.40 A.M			*Yes*			*No*	*No*
212.	1158/14	18	M	Single	RTA	Private	Treated	22.06.14 08.00 P.M	23.06.14 01.00 P.M			*Yes*			*No*	*No*
213.	1159/14	22	M	Married	Poisoning	Coolie	Treated	22.06.14 06.50 A.M	23.06.14 02.10 P.M				Yes		*No*	*No*
214.	1160/14	25	M	Married	RTA	Farmer	Treated	22.06.14 10.55 A.M	23.06.14 01.30 P.M				*Yes*		*No*	*No*
215.	1161/14	42	M	Married	RTA	Coolie	Treated	23.06.14 01.55 A.M	23.06.14 03.00 P.M			*Yes*			*No*	*No*
216.	1162/14	42	F	Married	RTA	House wife	Treated	23.06.14 06.15 A.M	23.06.14 02.40 P.M		Yes				*No*	*No*
217.	1163/14	45	M	Married	RTA	Driver	Treated	23.06.14 05.30 A.M	23.06.14 03.15 P.M		*Yes*				*No*	*No*
218.	1164/14	60	M	Married	RTA	Tailor	Brought dead	23.06.14 07.15 A.M	23.06.14 03.00 P.M		Yes				*No*	*No*
219.	1165/14	23	F	Single	Poisoning	Coolie	Treated	22.06.14 12.00 A.M	23.06.14 03.40 P.M					Yes	*No*	*No*
220.	1166/14	73	M	Married	RTA	Coolie	Treated	23.06.14 09.30 A.M	24.06.14 10.30 A.M				*Yes*		*No*	*No*
221.	1167/14	40	M	Married	Natural	Coolie	Brought dead	23.06.14 02.30 P.M	24.06.14 10.40 A.M			Yes			*No*	*No*
222.	1169/14	24	M	Single	Burns	Coolie	Brought dead	24.06.14 10.30 A.M	24.06.14 01.10 P.M	Yes					*No*	*No*
223.	1171/14	62	M	Married	RTA	Business	Treated	24.06.14 06.50 A.M	24.06.14 02.10 P.M		Yes				*No*	*No*
224.	1172/14	45	M	Married	Fall	Farmer	Treated	24.06.14 09.48 A.M	25.06.14 10.20 A.M				Yes		*No*	*No*
225.	1173/14	34	M	Married	Murder	Coolie	Treated	25.06.14 05.30 A.M	25.06.14 12.30 P.M		Yes				*No*	***Yes***
226.	1174/14	40	M	Married	RTA	Painter	Treated	24.06.14 08.15 A.M	25.06.14 01.10 P.M				Yes		*No*	*No*
227.	1175/14	54	M	Married	RTA	Coolie	Treated	24.06.14 04.00 P.M	25.06.14 01.40 P.M			*Yes*			*No*	*No*
228.	1176/14	38	M	Married	RTA	Mason	Treated	24.06.14 04.45 P.M	25.06.14 03.00 P.M			*Yes*			*No*	*No*

229.	1177/14	60	M	Married	RTA	Coolie	Treated	25.06.14 10.25 A.M	26.06.14 11.10 A.M				*Yes*		*No*	*No*
230.	1178/14	21	M	Single	Murder	Coolie	Treated	25.06.14 06.30 P.M	26.06.14 11.40 A.M			*Yes*			*No*	*No*
231.	1182/14	73	M	Married	RTA	Coolie	Treated	25.06.14 08.40 P.M	26.06,14 01.30 P.M			*Yes*			*No*	*No*
232.	1184/14	58	M	Married	Fall	Tailor	Treated	26.06.14 08.20 A.M	26.06.14 03.45 P.M		Yes				*No*	*No*
233.	1186/14	55	M	Married	RTA	Coolie	Brought dead	26.06.14 08.30 P.M	27.06.14 03.00 P.M			*Yes*			*No*	*No*
234.	1187/14	10	M	Single	RTA	Student	Brought dead	27.06.14 07.30 A.M	27.06.14 03.30 P.M		*Yes*				*No*	*No*
235.	1189/14	45	M	Married	RTA	Private	Treated	27.06.14 10.30 A.M	28.06.14 10.40 A.M				Yes		*No*	*No*
236.	1190/14	53	M	Married	RTA	Private	Treated	27.06.14 02.30 P.M	28.06.14 11.10 A.M			Yes			*No*	*No*
237.	1192/14	47	M	Married	Natural	Coolie	Brought dead	27.06.14 07.15 P.M	28.06.14 11.40 A.M			Yes			*No*	***Yes***
238.	1196/14	77	M	Married	TTA	Coolie	Brought dead	29.06.14 07.30 A.M	29.06.14 12.55 P.M	Yes					*No*	*No*
239.	1197/14	25	M	Single	Fall	Coolie	Treated	29.06.14 04.40 P.M	30.06.14 11.30 A.M			*Yes*			*No*	*No*
240.	1199/14	11	M	Single	RTA	Student	Treated	29.06.14 10.30 P.M	30.06.14 01.10 P.M			*Yes*			*No*	*No*
241.	1200/14	57	M	Married	Fall	Coolie	Treated	29.06.14 08.30 P.M	30.06.14 02.10 P.M			*Yes*			*No*	*No*
242.	1202/14	73	F	Married	Fall	Coolie	Treated	29.06.14 06.00 P.M	30.06.14 02.00 P.M			*Yes*			*No*	*No*
243.	1203/14	28	M	Married	RTA	Driver	Treated	30.06.14 06.45 A.M	30.06.14 03.10 P.M		*Yes*				*No*	*No*
244.	1204/14	40	M	Married	RTA	Private	Treated	30.06.14 03.40 A.M	30.06.14 03.30 P.M		Yes				*No*	*No*
245.	1205/14	47	M	Married	Poisoning	Painter	Treated	30.06.14 11.00 A.M	30.06.14 04.00 P.M	Yes					*No*	*No*
246.	1206/14	60	M	Married	RTA	Coolie	Treated	29.06.14 08.00 P.M	01.07.14 11.15 A.M					*Yes*	*No*	*No*

247.	1207/14	22	M	Single	RTA	Coolie	Treated	30.06.14 11.50 P.M	01.07.14 11.40 A.M		*Yes*				*No*	*No*
248.	1208/14	45	M	Married	RTA	Coolie	Treated	30.06.14 06.10 P.M	01.07.14 12 Noon			*Yes*			*No*	*No*
249.	1209/14	32	M	Married	Murder	Coolie	Treated	30.06.14 10.45 P.M	01.07.14 12.10 P.M			Yes			*No*	*No*
250.	1210/14	30	M	Married	RTA	Mason	Treated	30.06.14 03.50 P.M	01.07.14 12.40 P.M			Yes			*No*	*No*
251.	1211/14	35	M	Married	RTA	Coolie	Treated	30.06.14 04.25 P.M	01.07.14 01.30 P.M			Yes			*No*	*No*
252.	1212/14	54	M	Married	RTA	Farmer	Treated	30.06.14 09.50 A.M	01.07.14 01.30 P.M				Yes		*No*	*No*
253.	1214/14	65	M	Married	Natural	Coolie	Treated	28.06.14 12.30 P.M	01.07.14 02.00 P.M					*Yes*	*No*	*No*
254.	1216/14	45	F	Married	RTA	Coolie	Treated	01.07.14 06.30 A.M	01.07.14 04.30 P.M		Yes				*No*	*No*
255.	1217/14	22	M	Single	RTA	Private	Treated	01.07.14 12.30 P.M	02.07.14 11.10 A.M		*Yes*				*No*	*No*
256.	1218/14	50	M	Married	Fall	Coolie	Treated	01.07.14 10.55 P.M	02.07.14 11.40 A.M			*Yes*			*No*	*No*
257.	1219/14	58	M	Married	RTA	Coolie	Treated	01.07.14 05.40 A.M	02.07.14 12.10 P.M				*Yes*		*No*	*No*
258.	1220/14	35	M	Married	RTA	Coolie	Treated	01.07.14 07.20 A.M	02.07.14 12.30 P.M				*Yes*		*No*	*No*
259.	1221/14	36	M	Married	Hanging	Coolie	Treated	01.07.14 11.10 A.M	02.07.14 12.40 P.M				*Yes*		*No*	*No*
260.	1222/14	42		Married	RTA	Coolie	Treated	01.07.14 03.15 P.M	02.07.14 01.10 P.M			*Yes*			*No*	*No*
261.	1224/14	15	M	Single	RTA	Student	Brought dead	02.07.14 08.30 A.M	02.07.14 02.10 P.M	Yes					*No*	*No*
262.	1225/14	46	M	Married	RTA	Coolie	Treated	02.07.14 05.30 A.M	03.07.14 11.30 A.M				Yes		*No*	*No*
263.	1227/14	58	M	Married	RTA	Govt	Treated	02.07.14 04.15 P.M	03.07.14 11.40 A.M			*Yes*			*No*	*No*
264.	1228/14	72	M	Married	RTA	Private	Brought dead	02.07.14 12.20 P.M	03.07.14 11.30 A.M			Yes			*No*	*No*

265.	1229/14	25	F	Single	TTA	Student	Brought dead	02.07.14 02.10 P.M	03.07.14 12.20 P.M			*Yes*			*No*	*No*
266.	1230/14	70	M	Married	RTA	Coolie	Treated	02.07.14 03.30 P.M	03.07.14 01.00 P.M			*Yes*			*No*	*No*
267.	1231/14	22	M	Single	Fall	Painter	Brought dead	02.07.14 02.45 P.M	03.07.14 01.10 P.M			*Yes*			*No*	*No*
268.	1233/14	58	M	Married	RTA	Private	Treated	03.07.14 03.55 A.M	03.07.14 02.10 P.M		*Yes*				*No*	*No*
269.	1234/14	42	M	Married	Hanging	Fish man	Brought dead	03.07.14 11.00 A.M	03.07.14 03.30 P.M	Yes					*No*	***Yes***
270.	1235/14	36	M	Married	RTA	Coolie	Treated	03.07.14 06.25 A.M	03.07.14 04.30 P.M				Yes		*No*	*No*
271.	1236/14	30	M	Married	RTA	Coolie	Treated	03.07.14 02.35 P.M	04.07.14 11.15 A.M			Yes			*No*	*No*
272.	1238/14	27	M	Single	RTA	Coolie	Treated	03.07.14 11.00 A.M	04.07.14 12 Noon				Yes		*No*	*No*
273.	1239/14	44	M	Married	RTA	Private	Treated	03.07.14 09.00 P.M	04.07.14 12.30 P.M			Yes			*No*	*No*
274.	1240/14	50	M	Married	RTA	Farmer	Treated	03.07.14 06.10 A.M	04.07.14 01.30 P.M				Yes		*No*	*No*
275.	1241/14	8	F	Single	RTA	Student	Brought dead	04.07.14 01.25 P.M	04.07.14 03.00 P.M	Yes					*No*	*No*
276.	1242/14	65	M	Married	Natural	Coolie	Brought dead	04.07.14 06.55 A.M	04.07.14 03.30 P.M		Yes				*No*	*No*
277.	1244/14	26	F	Married	Murder	Sex worker	Brought dead	04.07.14 01.50 P.M	05.07.14 01.10 P.M			*Yes*			*No*	***Yes***
278.	1246/14	37	M	Married	Fall	Coolie	Treated	04.07.14 05.15 P.M	05.07.14 03.30 P.M			Yes			*No*	*No*
279.	1247/14	55	M	Married	RTA	Private	Treated	05.07.14 09.05 P.M	06.07.14 11.40 A.M			Yes			*No*	*No*
280.	1248/14	34	F	Married	Hanging	Private	Brought dead	05.07.14 07.05 P.M	06.07.14 12.30 P.M			Yes			*No*	*No*
281.	1249/14	48	M	Married	RTA	Coolie	Treated	06.07.14 04.30 A.M	06.07.14 01.30 P.M		Yes				*No*	*No*
282.	1252/14	52	M	Married	RTA	Coolie	Treated	06.07.14 09.30 P.M	07.07.14 12.30 P.M			Yes			*No*	*No*

283.	1253/14	52	M	Married	Poisoning	Driver	Treated	06.07.14 09.45 A.M	07.07.14 12.30 P.M				*Yes*		*No*	*No*
284.	1254/14	32	M	Single	Fall	Carpenter	Treated	06.07.14 09.10 A.M	07.07.14 03.10 P.M				*Yes*		*No*	*No*
285.	1255/14	22	M	Single	Fall	Carpenter	Treated	06.07.14 09.10 A.M	07.07.14 03.30 P.M				*Yes*		*No*	*No*
286.	1256/14	36	F	Married	Electrocut ion	Coolie	Treated	07.07.14 07.30A.M.	07.07.14 O4.00P.M		*Yes*				*No*	*No*
287.	1257/14	18	M	Single	RTA	Private	Treated	08.07.14 01.30 A.M	08.07.14 12.15 P.M		*Yes*				*No*	*No*
288.	1272/14	42	M	Married	Natural	Carpenter	Treated	08.07.14 06.00 A.M	08.07.14 02.40 P.M		*Yes*				*No*	*No*
289.	1274/14	45	M	Married	Snake Bite	Coolie	Treated	08.07.14 11.30 P.M	09.07.14 12 Noon			Yes			*No*	*No*
290.	1276/14	68	M	Married	Natural	Private	Treated	09.07.14 09.50 A.M	09.07.14 01.15 P.M	Yes					*No*	*No*
291.	1277/14	52	M	Married	Poisoning	Coolie	Treated	09.07.14 07.40 A.M	09.07.14 02.15 P.M		*Yes*				*No*	*No*
292.	1279/14	65	M	Married	Fall	Coolie	Treated	09.07.14 03.25 A.M	09.07.14 02.45 P.M		Yes				*No*	*No*
293.	1280/14	28	M	Married	RTA	Coolie	Treated	08.07.14 05.15 P.M	09.07.14 03.00 P.M			*Yes*			*No*	*No*
294.	1281/14	18	M	Single	TTA	Student	Treated	09.07.14 06.00 A.M	09.07.14 03.40 P.M		*Yes*				*No*	*No*
295.	1282/14	40	M	Married	RTA	Coolie	Treated	09.07.14 01.00 P.M	10.07.14 11.30 A.M			*Yes*			*No*	*No*
296.	1283/14	47	M	Married	RTA	Private	Treated	09.07.14 04.10 P.M	10.07.14 11.30 A.M			Yes			*No*	*No*
297.	1284/14	34	M	Married	Murder	Driver	Brought dead	09.07.14 06.45 P.M	10.07.14 12.30 P.M			Yes			*No*	***Yes***
298.	1285/14	65	F	Married	RTA	Coolie	Treated	10.07.14 05.30 A.M	10.07.14 01.00 P.M		Yes				*No*	*No*
299.	1287/14	34	M	Married	RTA	Driver	Treated	09.07.14 06.45 P.M	10.07.14 12.15 P.M			*Yes*			*No*	*No*
300.	1288/14	59	M	Married	Fall	Coolie	Treated	10.07.14 07.10 A.M	10.07.14 01.40 P.M		*Yes*				*No*	*No*

301.	1289/14	48	M	Married	Fall	Mason	Treated	09.07.14 05.25 P.M	10.07.14 02.00 P.M			*Yes*			*No*	*No*
302.	1290/14	53	M	Married	RTA	Farmer	Treated	09.07.14 03.00 P.M	10.07.14 02.10 P.M			*Yes*			*No*	*No*
303.	1291/14	53	M	Married	RTA	Farmer	Treated	09.07.14 03.00 P.M	10.07.14 02.15 P.M			*Yes*			*No*	*No*
304.	1292/14	28	F	Married	Hanging	House wife	Brought dead	09.07.14 11.35 P.M	10.07.14 04.30 P.M			Yes			*No*	*No*
305.	1293/14	59	F	Married	RTA	House wife	Treated	09.07.14 09.50 P.M	10.07.14 04.30 P.M			Yes			*No*	*No*
306.	1294/14	18	F	Single	Hanging	Coolie	Treated	10.07.14 04.25 P.M	11.07.14 11.30 A.M			Yes			*No*	*No*
307.	1297/14	49	M	Married	RTA	Coolie	Treated	10.07.14 10.30 P.M	11.07.14 01.30 P.M			Yes			*No*	*No*
308.	1298/14	73	M	Married	Snake Bite	Coolie	Treated	11.07.14 06.10 A.M	11.07.14 02.00 P.M		Yes				*No*	*No*
309.	1300/14	46	M	Married	Fall	Coolie	Treated	11.07.14 02.00 P.M	12.07.14 11.15 A.M			*Yes*			*No*	*No*
310.	1303/14	48	M	Married	Snake Bite	Coolie	Treated	11.07.14 04.10 P.M	12.07.14 01.40 P.M			*Yes*			*No*	*No*
311.	1304/14	24	M	Single	Poisoning	Coolie	Treated	11.07.14 03.30 P.M	12.07.14 02.40 P.M			*Yes*			*No*	*No*
312.	1305/14	58	M	Married	RTA	Private	Treated	12.07.14 12.30 A.M	12.07.14 04.00 P.M			Yes			*No*	*No*
313.	1308/14	35	F	Married	Natural	Coolie	Treated	12.07.14 07.35 P.M	13.07.14 02.00 P.M			*Yes*			*No*	*No*
314.	1309/14	61	M	Married	Fall	Broker	Brought dead	13.07.14 01.30 A.M	13.07.14 02.10 P.M			*Yes*			*No*	*No*
315.	1311/14	38	M	Married	TTA	Private	Brought dead	13.07.14 09.00 A.M	13.07.14 03.30 P.M		Yes				*No*	*No*
316.	1312/14	21	M	Single	RTA	Coolie	Treated	13.07.14 10.30 A.M	14.07.14 11.00 A.M				*Yes*		*No*	*No*
317.	1313/14	28	M	Single	Fall	Private	Treated	13.07.14 11.53 P.M	14.07.14 11.15 A.M			*Yes*			*No*	*No*
318.	1314/14	40	M	Married	Natural	Private	Treated	14.07.14 01.05 A.M	14.07.14 11.40 A.M		*Yes*				*No*	*No*

319.	1315/14	36	M	Married	TTA	Private	Brought dead	13.07.14 07.00 P.M	14.07.14 12 Noon			Yes			*No*	*No*
320.	1316/14	26	M	Single	RTA	Private	Brought dead	13.07.14 01.00 P.M	14.07.14 12.30 P.M			Yes			*No*	*No*
321.	1317/14	42	F	Married	Natural	House wife	Treated	13.07.14 05.30 P.M	14.07.14 12.15 P.M			*Yes*			*No*	*No*
322.	1318/14	65	M	Married	Poisoning	Coolie	Treated	13.07.14 12.45 P.M	14.07.14 01.10 P.M				*Yes*		*No*	*No*
323.	1319/14	25	M	Married	Electrocution	Coolie	Treated	13.07.14 08.00 P.M	14.07.14 01.30 P.M			*Yes*			*No*	*No*
324.	1320/14	23	M	Single	RTA	Driver	Treated	13.07.14 10.45 P.M	14.07.14 01.40 P.M			Yes			*No*	*No*
325.	1321/14	18	M	Single	Poisoning	Coolie	Treated	14.07.14 05.20 A.M	14.07.14 02.30 P.M		Yes				*No*	*No*
326.	1323/14	18	M	Single	RTA	Student	Treated	14.07.14 08.15 P.M	15.07.14 12.15 P.M			Yes			*No*	*No*
327.	1324/14	23	M	Married	RTA	Coolie	Treated	14.07.14 04.45 P.M	15.07.14 12.30 P.M			Yes			*No*	*No*
328.	1326/14	36	M	Married	Fall	Coolie	Treated	14.07.14 10.00 P.M	15.07.14 01.30 P.M			Yes			*No*	*No*
329.	1329/14	50	M	Married	RTA	Private	Treated	15.07.14 12.15 A.M	15.07.14 02.40 P.M			Yes			*No*	*No*
330.	1330/14	46	M	Married	Fall	Private	Treated	15.07.14 02.30 A.M	15.07.14 03.30 P.M		*Yes*				*No*	*No*
331.	1331/14	35	M	Married	Natural	Private	Treated	15.07.14 02.45 A.M	15.07.14 03.30 P.M		*Yes*				*No*	*No*
332.	1332/14	60	M	Married	Natural	Coolie	Treated	15.07.14 06.00 A.M	16.07.14 12.10 P.M				*Yes*		*No*	*No*
333.	1334/14	60	M	NK	Natural	NK	Treated	15.07.14 06.15 A.M	16.07.14 12.30 P.M				*Yes*		*No*	***Yes***
334.	1338/14	27	F	Married	Poisoning	House wife	Treated	15.07.14 03.00 P.M	16.07.14 04.30 P.M				*Yes*		*No*	*No*
335.	1340/14	66	M	Married	Fall	Fish man	Treated	16.07.14 07.15 P.M	17.07.14 11.30 A.M			*Yes*			*No*	*No*
336.	1341/14	32	M	Married	Hanging	Private	Treated	17.07.14 06.10 A.M	17.07.14 03.30 P.M		*Yes*				*No*	*No*

337.	1343/14	37	M	Married	RTA	Private	Treated	17.07.14 06.20 A.M	18.07.14 11.40 A.M				Yes		*No*	*No*
338.	1345/14	35	M	Single	RTA	Private	Treated	15.07.14 09.15 A.M	18.07.14 12.30 P.M					Yes	*No*	*No*
339.	1346/14	31	M	Married	RTA	Private	Treated	18.07.14 05.00 A.M	18.07.14 12.40 P.M		Yes				*No*	*No*
340.	1348/14	45	M	Single	Natural	Coolie	Treated	16.07.14 08.15 P.M	18.07.14 02.00 P.M					*Yes*	*No*	*No*
341.	1349/14	26	M	Single	TTA	Private	Brought dead	18.07.14 12.50 P.M	18.07.14 01.40 P.M	Yes					*No*	*No*
342.	1352/14	70	M	Married	Snake Bite	Coolie	Treated	18.07.14 12.15 P.M	19.07.14 11.15 A.M			*Yes*			*No*	*No*
343.	1353/14	57	M	Married	RTA	Govt	Treated	18.07.14 11.50 A.M	19.07.14 11.25 A.M			*Yes*			*No*	*No*
344.	1354/14	36	M	Single	RTA	Private	Treated	18.07.14 03.15 P.M	19.07.14 12.30 P.M			*Yes*			*No*	*No*
345.	1355/14	40	M	Married	Fall	Coolie	Treated	18.07.14 03.30 P.M	19.07.14 12.15 P.M			*Yes*			*No*	*No*
346.	1356/14	54	F	Married	Snake Bite	Coolie	Treated	18.07.14 06.50 P.M	19.07.14 12.40 P.M			*Yes*			*No*	*No*
347.	1358/14	29	M	Single	RTA	Private	Brought dead	18.07.14 04.45 P.M	19.07.14 02.00 P.M			*Yes*			*No*	*No*
348.	1359/14	38	M	Married	RTA	Coolie	Treated	18.07.14 01.00 P.M	19.07.14 03.00 P.M				*Yes*		*No*	*No*
349.	1360/14	51	F	Married	RTA	Private	Treated	19.07.14 06.45 P.M	20.07.14 10.30 A.M			Yes			*No*	*No*
350.	1363/14	24	M	Married	RTA	Coolie	Treated	20.07.14 01.00 A.M	20.07.14 01.30 P.M			*Yes*			*No*	*No*
351.	1366/14	33	M	Married	Murder	Butcher	Treated	21.07.14 05.45 A.M	21.07.14 11.40 A.M	Yes					*No*	*No*
352.	1367/14	12	F	Single	RTA	Student	Treated	20.07.14 03.45 P.M	21.07.14 01.15 P.M			*Yes*			*No*	*No*
353.	1368/14	52	M	Married	RTA	Coolie	Treated	20.07.14 11.25 A.M	21.07.14 01.45 P.M				*Yes*		*No*	*No*
354.	1369/14	35	M	Married	RTA	Driver	Treated	21.07.14 10.50 A.M	22.07.14 11.00 A.M				*Yes*		*No*	*No*

355.	1370/14	57	F	Married	RTA	Coolie	Treated	21.07.14 06.55 P.M	22.07.14 11.30 A.M			*Yes*			*No*	*No*
356.	1375/14	9	M	Single	Suspicious Death	Student	Treated	21.07.14 02.15 P.M	22.07.14 03.15 P.M				*Yes*		*No*	*No*
357.	1376/14	52	M	Single	Natural	Coolie	Treated	22.07.14 11.05 A.M	23.07.14 10.30 A.M			Yes			*No*	*No*
358.	1383/14	40	M	Married	Hanging	Coolie	Treated	23.07.14 08.50 P.M	24.07.14 01.10 P.M			*Yes*			*No*	*No*
359.	1384/14	60	M	Married	RTA	Coolie	Treated	24.07.14 05.30 A.M	24.07.14 01.40 P.M		*Yes*				*No*	*No*
360.	1387/14	54	M	Married	RTA	Coolie	Treated	25.07.14 01.40 A.M	25.07.14 12 Noon		*Yes*				*No*	*No*
361.	1388/14	19	M	Single	TTA	Coolie	Treated	24.07.14 07.20 A.M	25.07.14 12.10 A.M				*Yes*		*No*	*No*
362.	1391/14	70	F	Married	RTA	Farmer	Treated	24.07.14 12.10 P.M	25.07.14 02.40 P.M				*Yes*		*No*	*No*
363.	1398/14	72	M	Married	RTA	Private	Treated	25.07.14 05.30 P.M	26.07.14 11.15A.M			*Yes*			*No*	*No*
364.	1399/14	52	M	Married	RTA	Private	Treated	25.07.14 07.55 A.M	26.07.14 11.50 A.M				Yes		*No*	*No*
365.	1401/14	33	M	Married	Natural	Coolie	Treated	25.07.14 01.10 P.M	26.07.14 01.30 P.M				*Yes*		*No*	*No*
366.	1403/14	27	F	Married	Natural	Coolie	Treated	25.07.14 10.00 P.M	26.07.14 03.30 P.M			*Yes*			*No*	*No*
367.	1405/14	38	M	Married	Fall	Mason	Treated	25.07.14 09.30 P.M	26.07.14 03.30 P.M			*Yes*			*No*	*No*
368.	1406/14	35	M	Married	Natural	Coolie	Brought dead	25.07.14 10.45 P.M	26.07.14 04.00 P.M			*Yes*			*No*	*No*
369.	1408/14	42	M	Married	Poisoning	Coolie	Treated	25.07.14 06.55 P.M	26.07.14 04.00 P.M			*Yes*			*No*	*No*
370.	1409/14	23	M	Married	RTA	Coolie	Treated	26.07.14 03.45 P.M	27.07.14 11.00 A.M			*Yes*			*No*	*No*
371.	1410/14	42	M	Married	Natural	Coolie	Treated	25.07.14 07.15 P.M	27.07.14 12 Noon					*Yes*	*No*	*No*
372.	1411/14	65	M	Married	RTA	Coolie	Treated	26.07.14 05.15 P.M	27.07.14 12.30 P.M			*Yes*			*No*	*No*

373.	1412/14	09	M	Single	Natural	Kid	Brought dead	26.07.14 03.00 P.M	27.07.14 01.00 P.M			*Yes*			*No*	*No*
374.	1414/14	59	M	Married	RTA	Coolie	Treated	27.07.14 01.30 A.M	27.07.14 01.10 P.M		*Yes*				*No*	*No*
375.	1415/14	24	M	Married	Hanging	Coolie	Treated	27.07.14 06.50 A.M	27.07.14 02.00 P.M		*Yes*				*No*	*No*
376.	1416/14	49	M	Married	RTA	Coolie	Treated	27.07.14 01.30 A.M	27.07.14 01.40 P.M			*Yes*			*No*	*No*
377.	1418/14	65	M	Married	RTA	Coolie	Treated	27.07.14 10.30 P.M	28.07.14 11.30A.M			*Yes*			*No*	*No*
378.	1421/14	22	M	Single	RTA	Private	Treated	27.07.14 02.30 P.M	28.07.14 02.00 P.M			*Yes*			*No*	*No*
379.	1422/14	47	M	Married	Fall	Coolie	Treated	27.07.14 06.30 P.M	28.07.14 02.30 P.M			*Yes*			*No*	*No*
380.	1423/14	18	M	Single	RTA	Student	Treated	27.07.14 05.50 P.M	28.07.14 02.30 P.M			*Yes*			*No*	*No*
381.	1424/14	28	M	Married	Natural	Coolie	Treated	28.07.14 03.50A.M	28.07.14 02.40 P.M		*Yes*				*No*	*No*
382.	1426/14	55	F	Married	Drowning	Coolie	Treated	28.07.14 08.30 A.M	29.07.14 11.00A.M				*Yes*		*No*	*No*
383.	1427/14	46	M	Married	Fall	Plumber	Treated	28.07.14 12.10 P.M	29.07.14 11.40A.M			*Yes*			*No*	*No*
384.	1428/14	55	M	Married	Drowning	Farmer	Treated	28.07.14 04.50 P.M	29.07.14 01.00 P.M			*Yes*			*No*	*No*
385.	1429/14	42	M	Married	RTA	Driver	Treated	28.07.14 06.45 P.M	29.07.14 12.15 P.M			*Yes*			*No*	*No*
386.	1430/14	45	M	Married	Natural	Coolie	Treated	28.07.14 12.30.P.M	29.07.14 01.00 P.M				*Yes*		*No*	*No*
387.	1431/14	47	M	Married	Hanging	Coolie	Treated	28.07.14 11.45 P.M	29.07.14 01.15 P.M			*Yes*			*No*	*No*
388.	1433/14	35	F	Married	Snake Bite	House wife	Treated	29.07.14 07.30A.M	29.07.14 01.30P.M		29.07.14 01.30P.M	*Yes*			*No*	*No*
389.	1434/14	35	M	Married	RTA	Coolie	Treated	29.07.14 06.45A.M	30.07.14 10.30 A.M				*Yes*		*No*	*No*
390.	1437/14	71	M	Married	RTA	Private	Treated	30.07.14 06.40 A.M	30.07.14 02.00 P.M		*Yes*				*No*	*No*

391.	1438/14	7	F	Single	RTA	Student	Brought dead	29.07.14 04.20 P.M	30.07.14 03.30 P.M			Yes			*No*	*No*
392.	1440/14	63	F	Married	RTA	House wife	Treated	31.07.14 12.30 A.M	31.07.14 02.40 P.M			*Yes*			*No*	*No*
393.	1441/14	58	M	Married	RTA	Coolie	Treated	31.07.14 09.50 P.M	01.08.14 11.40 A.M			*Yes`*			*No*	*No*
394.	1442/14	36	M	Married	RTA	Mason	Treated	31.07.14 11.30 A.M	01.08.14 03.30 P.M				*Yes*		*No*	*No*
395.	1451/14	45	M	Married	Hanging	Coolie	Treated	01.08.14 02.45P.M	02.08.14 12.40P.M			Yes			*No*	*No*
396.	1453/14	59	M	Married	RTA	Private	Treated	02.08.14 09.45 A.M	02.08.14 04.15 P.M		*Yes*				*No*	*No*
397.	1454/14	56	M	Married	RTA	Private	Treated	03.08.14 03.15 A.M	03.08.14 11.15 A.M		*Yes*				*No*	*No*
398.	1455/14	75	F	Married	RTA	House wife	Treated	02.08.14 05.30 P.M	03.08.14 11.45 P.M			*Yes*			*No*	*No*
399.	1456/14	21	M	Single	RTA	Cleaner	Brought dead	03.08.14 04.30 P.M	04.08.14 10.30 A.M			*Yes*			*No*	***Yes***
400.	1457/14	59	M	Married	RTA	House wife	Brought dead	03.08.14 01.20 P.M	04.08.14 10.40 A.M			*Yes*			*No*	*No*
401.	1458/14	28	M	Single	RTA	Driver	Treated	04.08.14 02.30 A.M	04.08.14 11.00 A.M		*Yes*				*No*	*No*
402.	1459/14	50	M	Married	Natural	Coolie	Treated	02.08.14 12.30 A.M	04.08.14 11.30 A.M					*Yes*	*No*	*No*
403.	1461/14	26	M	Married	RTA	Coolie	Treated	04.08.14 06.05 A.M	04.08.14 12.30 P.M		Yes				*No*	*No*
404.	1462/14	45	M	Married	Natural	Private	Treated	04.08.14 08.30 A.M	04.08.14 02.00 P.M	Yes					*No*	*No*
405.	1463/14	46	M	Married	RTA	Private	Treated	04.08.14 02.00 A.M	04.08.14 03.30 P.M			*Yes*			*No*	*No*
406.	1464/14	34	M	Married	RTA	Coolie	Treated	04.08.14 07.15 A.M	04.08.14 04.30 P.M		Yes				*No*	*No*
407.	1465/14	42	M	Married	TTA	Private	Treated	02.08.14 07.45 A.M	05.08.14 10.30 A.M					*Yes*	*No*	*No*
408.	1468/14	21	M	Married	RTA	Coolie	Treated	04.08.14	05.08.14				*Yes*		*No*	*No*

								12.15 P.M	12.40 P.M							
409.	1469/14	27	M	Single	Hanging	Driver	Treated	05.08.14 03.00 A.M	05.08.14 01.15 P.M		*Yes*				*No*	*No*
410.	1474/14	54	M	Married	Fall	Private	Treated	05.08.14 07.25 A.M	05.08.14 04.00 P.M		*Yes*				*No*	*No*
411.	1475/14	43	M	Married	RTA	Private	Treated	04.08.14 09.25 P.M	05.08.14 04.15 P.M			*Yes*			*No*	*No*
412.	1476/14	38	M	Married	Poisoning	Coolie	Treated	04.08.14 01.15 P.M	05.08.14 04.10 P.M				*Yes*		*No*	*No*
413.	1477/14	43	M	Married	RTA	Private	Brought dead	04.08.14 07.45 P.M	05.08.14 04.30 P.M			*Yes*			*No*	*No*
414.	1480/14	53	M	Single	Fall	Private	Treated	05.08.14 07.00 A.M	06.08.14 11.30 A.M				*Yes*		*No*	*No*
415.	1481/14	17	M	Single	RTA	Student	Treated	06.08.14 05.40 A.M	06.08.14 01.40 P.M		*Yes*				*No*	*No*
416.	1483/14	44	M	Married	RTA	Driver	Treated	06.08.14 12.15 P.M	07.08.14 11.30 A.M				*Yes*		*No*	*No*
417.	1484/14	24	M	Married	Murder	Coolie	Treated	06.08.14 09.30 A.M	07.08.14 12.30 P.M				Yes		*No*	*No*
418.	1486/14	68	M	Married	Natural	Coolie	Treated	06.08.14 03.30 P.M	07.08.14 02.00 P.M			*Yes*			*No*	*No*
419.	1488/14	42	M	Married	Fall	Coolie	Treated	07.08.14 12.05 A.M	07.08.14 03.30 P.M		*Yes*				*No*	*No*
420.	1489/14	55	M	Married	Natural	Coolie	Treated	07.08.14 05.30 A.M	07.08.14 03.30 P.M		*Yes*				*No*	*No*
421.	1490/14	24	M	Single	Poisoning	Govt	Treated	07.08.14 06.00 A.M	07.08.14 03.40 P.M		*Yes*				*No*	*No*
422.	1491/14	60	F	Married	RTA	House wife	Brought dead	07.08.14 08.40 A.M	08.08.14 11.00 A.M				*Yes*		*No*	*No*
423.	1492/14	37	M	Married	RTA	Farmer	Brought dead	07.08.14 09.00 A.M	08.08.14 11.00 A.M				Yes		*No*	*No*
424.	1494/14	23	F	Married	RTA	Coolie	Treated	07.08.14 09.45 A.M	08.08.14 01.00 P.M				*Yes*		*No*	*No*
425.	1496/14	25	M	Single	RTA	Coolie	Treated	07.08.14 03.50 P.M	08.08.14 02.30 P.M			*Yes*			*No*	*No*
426.	1498/14	22	M	Single	TTA	Student	Brought	09.08.14	09.08.14	Yes					*No*	*No*

							dead	08.00 A.M	11.00 A.M							
427.	1499/14	45	F	Married	TTA	Merchant	Brought dead	08.08.14 09.45 A.M	09.08.14 11.30 A.M				*Yes*		*No*	***Yes***
428.	1500/14	47	M	Married	Natural	Private	Treated	08.08.14 06.15 P.M	09.08.14 11.00 A.M			*Yes*			*No*	*No*
429.	1502/14	50	M	Married	RTA	Coolie	Treated	08.08.14 10.45 P.M	09.08.14 12.30 P.M			*Yes*			*No*	*No*
430.	1503/14	26	M	Single	RTA	Private	Brought dead	08.08.14 03.30 P.M	09.08.14 01.00 P.M			*Yes*			*No*	*No*
431.	1505/14	42	M	Married	RTA	Driver	Treated	09.08.14 05.00 A.M	09.08.14 03.00 P.M		*Yes*				*No*	*No*
432.	1506/14	29	M	Married	Natural	Coolie	Treated	09.08.14 08.30 A.M	09.08.14 03.15 P.M		*Yes*				*No*	*No*
433.	1507/14	14	M	Single	Scorpion Bite	Student	Brought dead	08.08.14 08.05 P.M	09.08.14 03.40 P.M			*Yes*			*No*	*No*
434.	1508/14	59	M	Married	RTA	Private	Treated	09.08.14 05.10 P.M	10.08.14 10.15 A.M			*Yes*			*No*	*No*
435.	1509/14	20	M	Single	RTA	Private	Treated	09.08.14 07.50 A.M	10.08.14 11.00 A.M				*Yes*		*No*	*No*
436.	1510/14	52	F	Married	RTA	House wife	Treated	10.08.14 02.30 A.M	10.08.14 11.30 A.M		*Yes*				*No*	*No*
437.	1511/14	22	M	Single	Natural	Private	Treated	10.08.14 07.15 A.M	10..08.14 01.00 P.M	Yes					*No*	*No*
438.	1512/14	70	M	Married	Fall	Coolie	Treated	10.08.14 07.40 A.M	10.08.14 03.30 P.M		*Yes*				*No*	*No*
439.	1513/14	45	M	Married	Poisoning	Coolie	Treated	10.08.14 07.25 P.M	11.08.14 01.00 P.M			*Yes*			*No*	*No*
440.	1514/14	23	M	Single	Accident	Coolie	Treated	09.08.14 02.30 P.M	11.08.14 01.15 P.M					*Yes*	*No*	*No*
441.	1516/14	45	M	Married	Poisoning	Coolie	Treated	11.08.14 02.25 P.M	12.08.14 12.30 P.M			*Yes*			*No*	*No*
442.	1517/14	24	M	Married	RTA	Private	Treated	10.08.14 10.45 P.M	12.08.14 12.30 P.M					*Yes*	*No*	*No*
443.	1518/14	61	M	Married	RTA	Private	Treated	11.08.14 11.20 P.M	12.08.14 12.40 P.M			Yes			*No*	*No*
444.	1520/14	40	M	Married	RTA	Painter	Treated	12.08.14	12.08.14		*Yes*				*No*	*No*

								04.30 A.M	01.15 P.M							
445.	1521/14	56	M	Married	Murder	Coolie	Treated	12.08.14 07.00 A.M	12.08.14 03.40 P.M		*Yes*				*No*	*No*
446.	1522/14	40	M	Married	RTA	Coolie	Treated	12.08.14 11.00 P.M	13.08.14 11.30 A.M			*Yes*			*No*	*No*
447.	1523/14	62	M	Married	Hanging	Private	Treated	12.08.14 01.45 P.M	13.08.14 11.30 A.M			*Yes*			*No*	*No*
448.	1524/14	22	M	Single	TTA	Coolie	Treated	12.08.14 06.50 P.M	13.08.14 12.30 P.M			*Yes*			*No*	*No*
449.	1526/14	22	M	Single	RTA	Coolie	Treated	13.08.14 05.20 A.M	13.08.14 01.40 A.M		*Yes*				*No*	*No*
450.	1527/14	28	M	Single	Fall	Coolie	Treated	12.08.14 10.00 A.M	13.08.14 02.30 P.M				*Yes*		*No*	*No*
451.	1528/14	42	M	Married	RTA	Coolie	Treated	12.08.14 10.20 P.M	13.08.14 02.40 P.M			*Yes*			*No*	*No*
452.	1529/14	30	M	Single	RTA	Coolie	Treated	12.08.14 06.45 P.M	13.08.14 03.30 P.M			*Yes*			*No*	*No*
453.	1530/14	60	M	Married	RTA	Painter	Treated	13.08.14 05.15 A.M	13.08.14 03.30 P.M		*Yes*				*No*	*No*
454.	1531/14	80	M	Married	Natural	Coolie	Treated	13.08.14 02.00 P.M	14.08.14 01.00 P.M			*Yes*			*No*	*No*
455.	1533/14	40	M	Married	Natural	Merchant	Treated	13.08.14 09.20 P.M	14.08.14 01.30 P.M			*Yes*			*No*	*No*
456.	1534/14	50	M	Married	RTA	Coolie	Brought dead	13.08.14 06.10 P.M	14.08.14 02.00 P.M			Yes			*No*	*No*
457.	1535/14	30	M	Married	RTA	Driver	Treated	14.08.14 09.30 A.M	15.08.14 10.15 A.M				*Yes*		*No*	*No*
458.	1536/14	30	M	Single	Poisoning	Coolie	Treated	14.08.14 09.00 A.M	15.08.14 11.30 A.M				*Yes*		*No*	*No*
459.	1537/14	34	M	Single	Poisoning	Coolie	Treated	14.08.14 10.50 P.M	15.08.14 12.10 P.M			*Yes*			*No*	*No*
460.	1538/14	45	M	Married	Natural	Coolie	Treated	14.08.14 09.30 P.M	15.08.14 01.30 P.M			*Yes*			*No*	*No*
461.	1539/14	39	M	Married	Fall	Coolie	Treated	14.08.14 09.45 P.M	15.08.14 02.30 P.M			*Yes*			*No*	*No*
462.	1540/14	27	M	Single	Poisoning	Coolie	Treated	14.08.14	15.08.14			*Yes*			*No*	*No*

								02.40 P.M	02.30 P.M							
463.	1541/14	60	M	Married	Fall	Private	Brought dead	15.08.14 08.20 A.M	15.08.14 02.30 P.M		*Yes*				*No*	*No*
464.	1542/14	68	M	Married	Natural	Coolie	Treated	15.08.14 08.50 A.M	15.08.14 03.00 P.M		*Yes*				*No*	*No*
465.	1543/14	26	M	Married	Electrocution	Coolie	Treated	15.08.14 03.00 A.M	15.08.14 03.30 P.M			*Yes*			*No*	*No*
466.	1575/14	48	M	Married	TTA	Private	Treated	20.08.14 12.50 P.M	20.08.14 02.30 P.M	Yes					*No*	*No*
467.	1620/14	2	F	Single	Poisoning	Kid	Treated	27.08.14 02.45 P.M	27.08.14 04.00 P.M	Yes					*No*	*No*
468.	1622/14	43	M	Single	Murder	Lawyer	Brought dead	28.08.14 03.22 A.M	28.08.14 11.40 A.M		*Yes*				*No*	*No*
469.	1625/14	25	M	Married	RTA	Private	Treated	28.08.14 04.30 P.M	29.08.14 12.30 P.M			*Yes*			*No*	*No*
470.	1628/14	51	M	Married	RTA	Coolie	Treated	28.08.14 03.40 P.M	29.08.14 03.00 P.M			*Yes*			*No*	*No*
471.	1629/14	6 months	M	Single	Fall	Kid	Treated	28.08.14 09.20 A.M	29.08.14 04.30 P.M					*Yes*	*No*	*No*
472.	1652/14	35	M	Single	RTA	Driver	Treated	31.08.14 10.15 P.M	01.09.14 03.30 P.M			*Yes*			*No*	*No*
473.	1656/14	60	M	Married	RTA	Private	Treated	02.09.14 04.00 A.M	02.09.14 11.30 A.M		*Yes*				*No*	*No*
474.	1657/14	40	M	Married	TTA	Merchant	Treated	01.09.14 07.20 P.M	02.09.14 12.30 P.M			Yes			*No*	*No*
475.	1658/14	78	M	Married	RTA	Coolie	Treated	01.09.14 03.15 P.M	02.09.14 01.30 P.M			Yes			*No*	*No*
476.	1659/14	64	F	Married	RTA	Merchant	Treated	02.09.14 01.50 A.M	02.09.14 01.30 P.M		*Yes*				*No*	*No*
477.	1660/14	50	F	Married	RTA	House wife	Treated	02.09.14 01.50 A.M	02.09.14 02.40 P.M			Yes			*No*	*No*
478.	1662/14	49	M	Married	RTA	Coolie	Treated	02.09.14 11.30 A.M	02.09.14 04.00 P.M	Yes					*No*	*No*
479.	1663/14	57	M	Married	RTA	Coolie	Treated	02.09.14	03.09.14			Yes			*No*	*No*

								02.45 P.M	11.00 A.M							
480.	1664/14	16	M	Single	Drowning	Student	Treated	02.09.14 11.15 A.M	03.09.14 11.00 A.M			Yes			*No*	*No*
481.	1665/14	35	M	Married	RTA	Coolie	Treated	03.09.14 12.40 A.M	03.09.14 02.00 P.M			Yes			*No*	*No*
482.	1666/14	21	M	Single	TTA	Student	Treated	03.09.14 07.15 A.M	03.09.14 04.00 P.M		*Yes*				*No*	*No*
483.	1667/14	32	M	Single	TTA	Coolie	Treated	03.09.14 04.50 P.M	04.09.14 11.00 A.M			Yes			*No*	*No*
484.	1668/14	50	M	Married	RTA	Govt	Treated	03.09.14 10.40 A.M	04.09.14 11.30 A.M				*Yes*		*No*	*No*
485.	1669/14	32	M	Married	RTA	Farmer	Brought dead	03.09.14 07.55 P.M	04.09.14 11.30 A.M			Yes			*No*	*No*
486.	1671/14	42	F	Married	Murder	Coolie	Treated	03.09.14 10.20 A.M	04.09.14 01.00 P.M				*Yes*		*No*	*No*
487.	1672/14	40	M	Married	Fall	Coolie	Treated	03.09.14 11.45 A.M	04.09.14 01.30 P.M				*Yes*		*No*	*No*
488.	1677/14	25	M	Married	TTA	Private	Brought dead	05.09.14 05.00 A.M	05.09.14 12.30 P.M		Yes				*No*	*No*
489.	1678/14	49	M	Married	Fall	Govt	Treated	04.09.14 04.15 P.M	05.09.14 03.00 P.M			Yes			*No*	*No*
490.	1681/14	25	M	Single	RTA	Private	Treated	05.09.14 01.15P.M	06.09.14 10.15 A.M			Yes			*No*	*No*
491.	1683/14	60	F	Married	RTA	Private	Brought dead	04.09.14 06.10 A.M	06.09.14 01.00 P.M					*Yes*	*No*	*No*
492.	1684/14	40	F	NK	TTA	NK	Treated	05.08.14 05.30 P.M	06.09.14 01.30 P.M			Yes			*No*	*No*
493.	1685/14	55	M	Single	Electrocution	Coolie	Treated	06.09.14 12.50 A.M	06.09.14 01.40 P.M			Yes			*No*	*No*
494.	1687/14	67	F	Married	Natural	Coolie	Treated	05.09.14 10.30 P.M	06.09.14 02.30 P.M			Yes			*No*	*No*
495.	1688/14	62	M	Married	RTA	Coolie	Treated	05.09.14 10.15 P.M	06.09.14 03.30 P.M			Yes			*No*	*No*
496.	1689/14	26	M	Single	Fall	Mechanic	Treated	06.09.14 09.30 A.M	07.09.14 11.00 A.M				*Yes*		*No*	*No*
497.	1690/14	46	M	Married	Natural	Coolie	Treated	06.09.14	07.09.14			Yes			*No*	***Yes***

								05.55 P.M	11.50 A.M							
498.	1691/14	39	M	Married	Industrial accident	Coolie	Treated	06.09.14 10.05 P.M	07.09.14 12.45 P.M			Yes			*No*	*No*
499.	1694/14	45	M	Married	Poisoning	Driver	Treated	07.09.14 08.00 A.M	08.09.14 10.45 A.M				*Yes*		*No*	*No*
500.	1695/14	45	M	Married	Electrocution	Coolie	Treated	07.09.14 08.30 A.M	08.09.14 11.00 A.M				*Yes*		*No*	*No*
501.	1696/14	45	M	Married	Fall	Coolie	Treated	07.09.14 12.15 A.M	08..09.14 03.30 P.M					*Yes*	*No*	*No*
502.	1699/14	70	M	Married	Poisoning	Coolie	Treated	08.09.14 12.10 P.M	09.09.14 11.40 A.M			Yes			*No*	*No*
503.	1703/14	38	M	Married	Natural	Coolie	Treated	08.09.14 08.20 A.M	09.09.14 01.30 P.M				*Yes*		*No*	*No*
504.	1707/14	26	M	Married	RTA	Coolie	Treated	08.09.14 06.00 A.M	09.09.14 02.30 P.M				*Yes*		*No*	*No*
505.	1709/14	30	M	Single	TTA	Coolie	Treated	06.09.14 03.30 P.M	10.09.14 12.15 P.M					*Yes*	*No*	*No*
506.	1712/14	45	M	Married	RTA	Coolie	Treated	09.09.14 01.10 P.M	10.09.14 01.30 P.M				*Yes*		*No*	*No*
507.	1713/14	75	F	Married	Poisoning	House wife	Treated	10.09.14 01.15 A.M	10.09.14 03.00 P.M			Yes			*No*	*No*
508.	1714/14	34	M	Married	RTA	Coolie	Treated	10.09.14 12.45 A.M	10.09.14 04.00 P.M			Yes			*No*	*No*
509.	1715/14	43	M	Married	RTA	Private	Treated	10.09.14 12.13 P.M	11.09.14 10.40 A.M			Yes			*No*	*No*
510.	1716/14	50	M	Married	RTA	Govt	Treated	10.09.14 03.55 P.M	11.09.14 11.00 A.M			Yes			*No*	*No*
511.	1718/14	50	F	Married	Natural	House wife	Treated	10.09.14 07.00 P.M	11.09.14 11.40 A.M			Yes			*No*	*No*
512.	1719.14	64	F	Married	RTA	Coolie	Treated	10.09.14 11.00 P.M	11.09.14 02.00 P.M			Yes			*No*	*No*
513.	1720/14	42	M	Single	RTA	Coolie	Treated	10.09.14 11.15 A.M	11.09.14 02.15 P.M				Yes		*No*	*No*
514.	1721/14	80	F	Married	Poisoning	House wife	Treated	10.09.14 06.15 P.M	11.09.14 02.00 P.M			Yes			*No*	*No*
515.	1722/14	60	M	Married	RTA	Coolie	Treated	10.09.14	11.09.14			Yes			*No*	*No*

								04.30 P.M	02.30 P.M							

Abbreviations:

1. **P.M No – Post mortem number**
2. **M – Male**
3. **F – Female**
4. **RTA – Road Traffic Accident**
5. **TTA – Train Traffic Accident**
6. **NK – Not Known**
7. **HBV – Hepatitis B Virus**
8. **HBsAg – Hepatitis B Surface antigen**